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Archives OF OTOLARYNGOLOGY

MAY 1956

VOLUME 63 NUMBER 5

External Otitis

Henry F. Brown

Astrocytomas of the Nose

Rogelio del Valle

Laryngoesophagus

Max L. Som

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Deafness and Vertigo from Head Injury

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Facial Paralysis and Muscle-Relaxant Anesthesia

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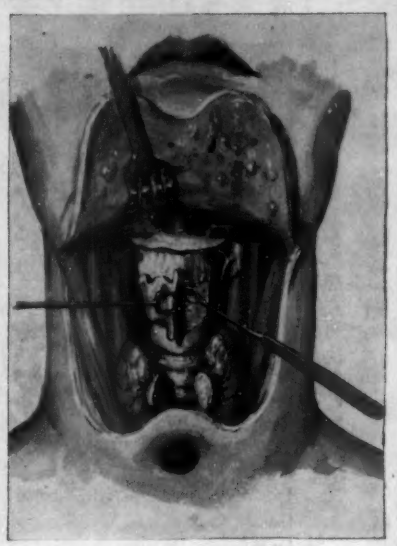

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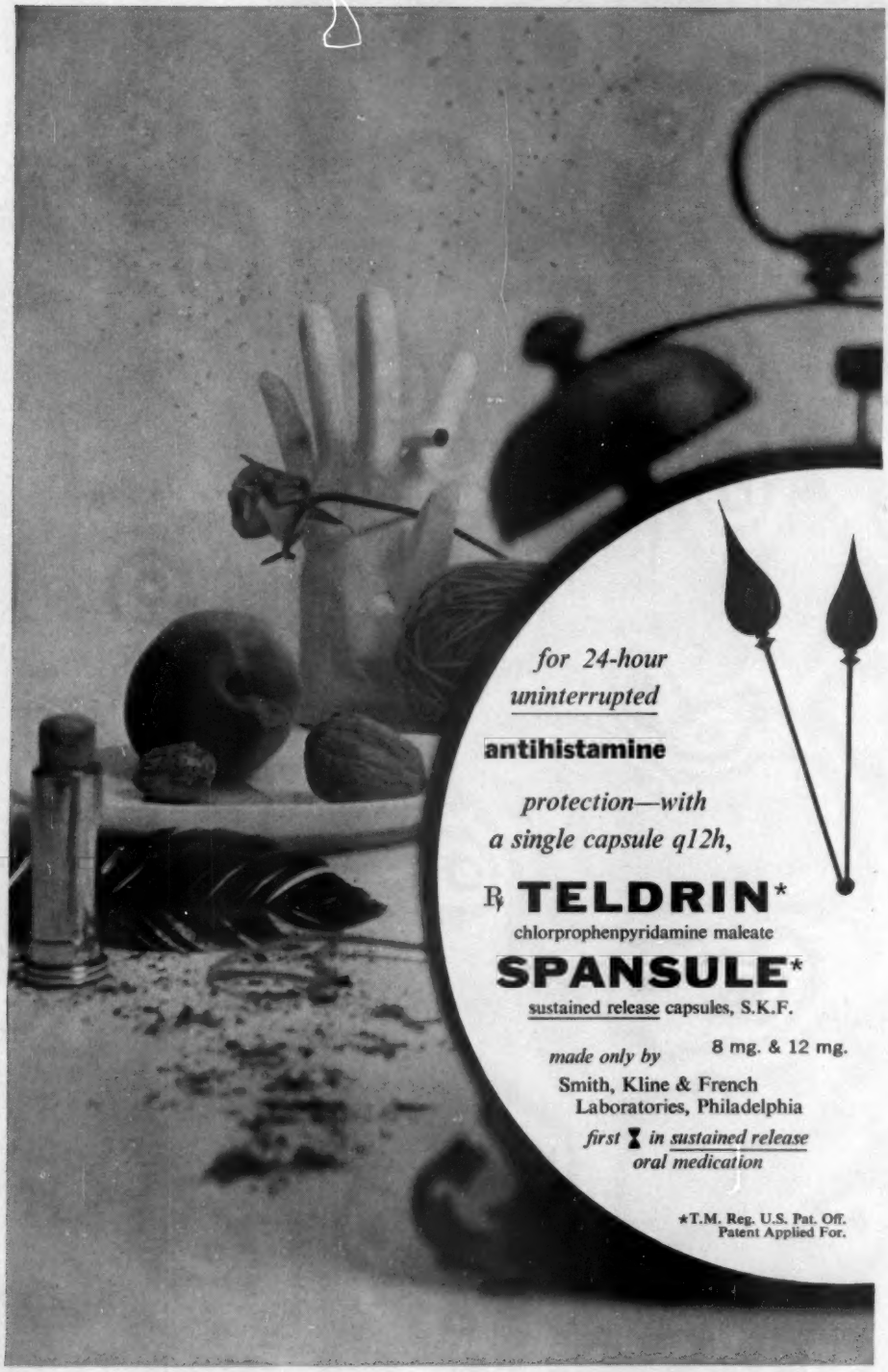
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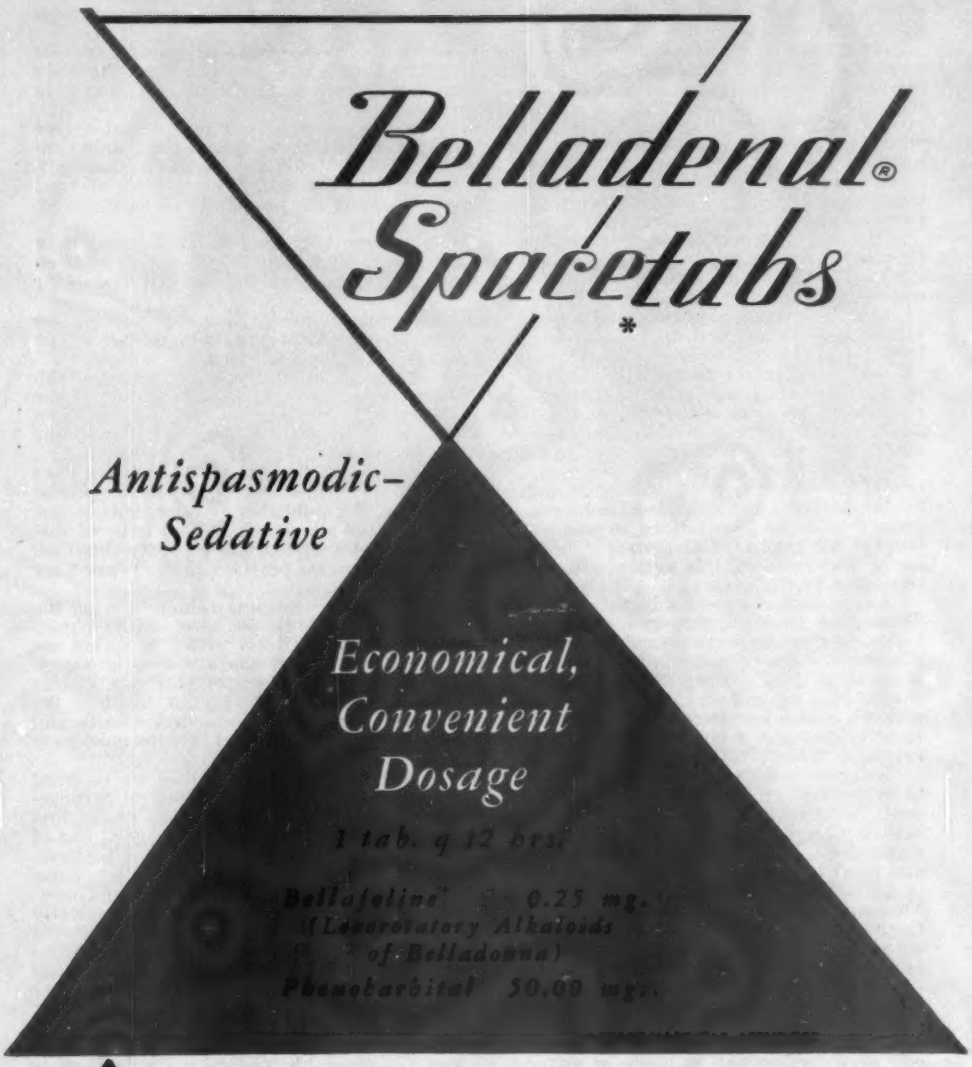

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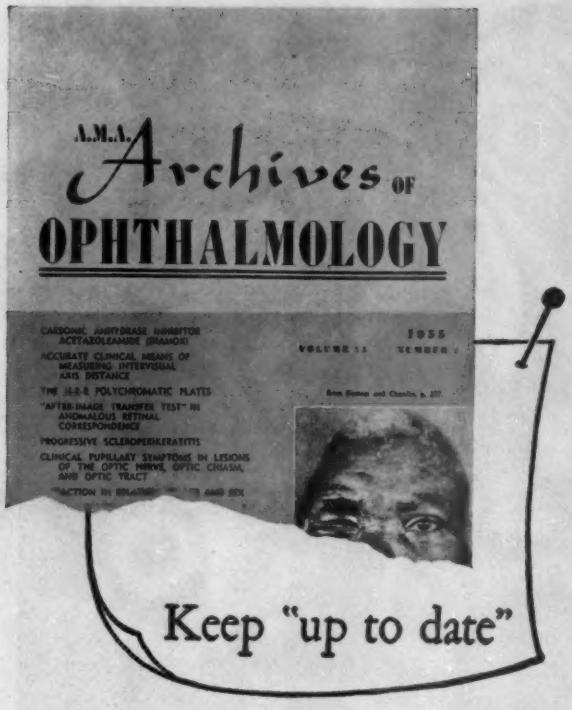

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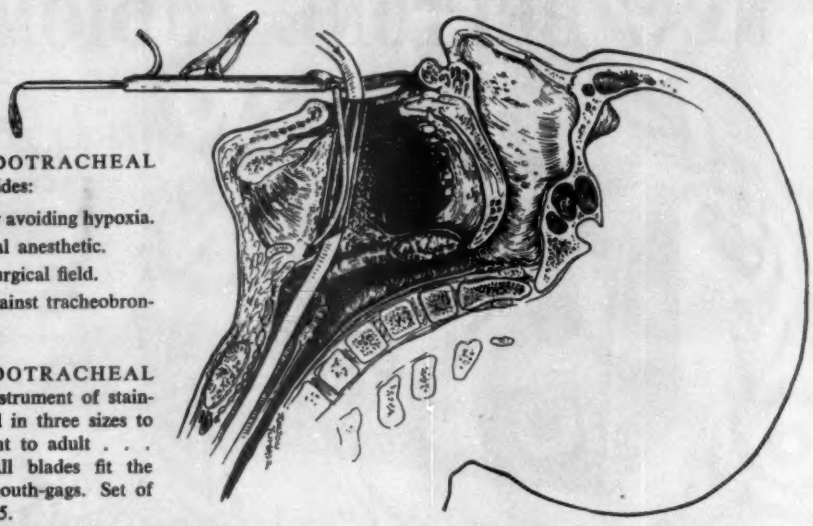

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External Otitis

A Method of Therapy and Prophylaxis

HENRY E. BRANCA, M.D., Fort Pierce, Fla.

Recent investigations into the etiology and pathogenesis of external otitis have brought about a more rational method of therapy and possibly prophylaxis. Studies of the bacteriology and mycology of external otitis have been going on for many years. It has been only during the past few years, however, that interest has been directed toward explaining why this condition is so characteristically recurrent. The antibiotics and their peculiar bacterial spectra of activity have changed our mode of thinking. When we are confronted with an infectious process we now ask ourselves the following: What is the organism? Is it pathogenic, and to what antibiotic is it sensitive? Since we have been armed with this information pathogenic organisms have become an easy prey to our drugs—a far cry from the practice of medicine several years ago.

All the recent investigations have uniformly reported that the vast majority of cases of external otitis are due to the Pseudomonas bacillus. The hemolytic Staphylococcus aureus is also a causative but less frequent factor.* The fungi have consistently lost support as a common offender in external otitis.† Molds as pathogens are reported in less than 10% of all cases. Seborrhea and psoriasis, while infrequent, should be mentioned only so that they not be ignored as possible factors.

Fortified with the knowledge of the causative organisms, investigators have tried to find explanations for the other characteristics of this disease. All agree that this is a condition prevalent in the hot, humid climates.‡ One writer significantly and appropriately heads his article "The Hot Weather Ear."[11] Senturia [12] offers a rational explanation for the occurrence of external otitis in the hot, humid months of the year. Most authors simply explain the hot climate relationship by saying that such an environment favors the growth of bacteria and molds.

There have been extensive studies of the pH factor of the external ear. Fabricant [13] found the pH of the normal ear canal to be on the acid side. The skin surface is covered by an "acid mantle" that defends against bacterial and fungicidal invasion. The "acid mantle" is due to the cornified horny cells and secretions of the sebaceous and apocrine glands. Bernstein [14] found the highest degree of alkalinity in fissures present in eczematous processes. Gill [15] showed that in 90% of cases of bacterial and fungoid external otitis the discharge was alkaline. He preferred drugs with a pH on the acid side. Ochs § demonstrated the effectiveness of the acid pH factor by obtaining favorable results with household vinegar.

The role ear cerumen plays in external otitis has been and is under investigation. The absence of cerumen in established cases

Accepted for publication Dec. 27, 1955.

* References 1 to 5.

† References 4 to 7.

‡ References 8 to 10.

§ References 16 and 17.

is well known.‖ It has already been mentioned that the secretions of the ear-canal glands are acid in pH and inhibit growth of bacteria and molds. Biopsies ¶ of skin of ears having external otitis show destruction of the normal glandular structure. This gives histologically the reason for the absence of ear wax. The logical channel for further investigation therefore was and is the study of the chemical nature of cerumen.#

If cerumen can be artificially synthesized, then external otitis may be controlled by a form of substitutive therapy. We are all eagerly awaiting the results of present investigations along these lines.

Several years ago I began keeping a graph of the seasonal incidence of external otitis. While it has been well established that this condition occurs in the summer months, I do not recall ever seeing a statistical report showing the incidence of external otitis for all the seasons of the year. While one realizes that a graph such as this applies only to one's immediate area of the nation, it would be interesting to see what similar studies elsewhere would reveal. This basic knowledge may someday give a clue to a better understanding of otitis externa.

Several years ago [25] I reported on some observations and drew some conclusions regarding external otitis. I was impressed with the variation in the clinical picture in the same patient over a long period of time. A patient may at one time present furunculosis, later a clear serous discharge, later "green pus," later fungus, and, finally, a dry, scaly type of eczematoid lesion. In the report on a series of cases it was emphasized that even though all semblance of infection was eradicated recurrences were the rule. The pruritus remaining in a healed ear soon brought on reinfection by the incessant scratching, and the cycle of events would occur over and over again. I offered the theory of atrophy of the ceruminous glands as the basic factor in external otitis. We otologists were not treating the specific disease but were simply removing the secondary invaders. I indicated at the time that I did not know if any biopsies of the skin of the ear canal had been made.

In view of the recent investigations * on the histologic studies of the skin of the ear canal—in both the normal and the abnormal—and the success with the corticosteroids in the treatment of external otitis, great strides forward have been made in the comprehension of this disease. Since it is now established that there is atrophy of the ceruminous glands and that trauma by scratching introduces the secondary infection,† the following clinical impressions may be formulated. External otitis has its beginning in atrophy of the glandular components of the skin of the ear canal. (The cause of this is unknown or given as infection, edema, or obstruction.) The dryness of the canal initiates pruritus. The absence of normal secretions reduces the barriers to fungicidal or bacterial invasion.[26] The pruritus introduces the trauma element, which, in turn, brings on invasion by patho-

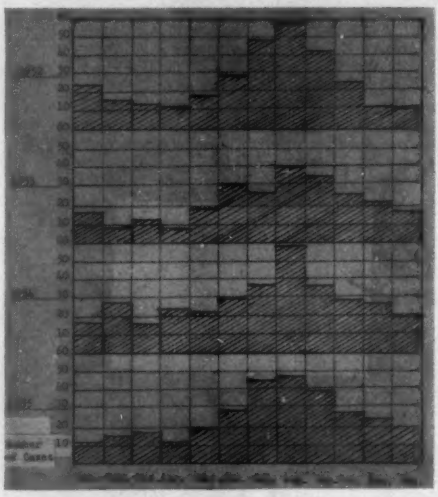

Fig. 1.—Month-by-month incidence of external otitis in southern Florida. The uniformity of incidence over a four-year period is evident.

‖ References 2, 18 to 21.

¶ References 22 and 23.

References 11, 23, and 24.

* References 22 and 23.

† References 11, 24, and 25.

genic organisms, and a full-blown case of external otitis is thus established. The more prolonged the infection the more the destruction of the glandular element occurs.

Now let us see how the present mode of therapy is applied. For the atrophy of the glands wax substitutes are now under investigation [23] and their use may become the future method of treatment. Since at present the disease can not be controlled at this level, we may try to do so at the stage of pruritus (preinfectious?). Here the corticosteroids ‡ have worked brilliantly, for without scratching secondary infection may not occur. The corticosteroid is combined with an antibiotic

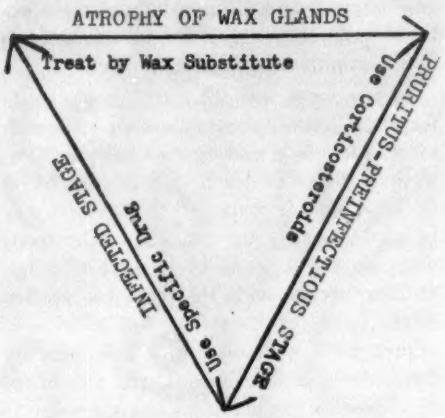

Fig. 2.—The cycle of external otitis.

to further inhibit bacteria. If the external otitis is well established and presents obvious invasion by organisms, then these must be removed first by proper chemotherapy, fungicides, or antibiotics. Thus the cycle of events may be broken by (1) a wax substitute, (2) an antipruritic, and (3) specific drugs. This scheme of events may be presented diagrammatically (Fig. 2).

Many cases of external otitis usually first come to us with furunculosis. Many of these patients give a history of swimming, but as many do not give this history. There often is some itching, serous drainage, or sense of fullness before the pain starts. These are the cases that several years ago gave us so much difficulty in controlling and in preventing recurrences: After getting over one bout they soon would develop another. These are also the cases that because of the failure to cure early developed "chronic external otitis." It now becomes evident that because of the recurrent attacks the glandular structure of the ear was destroyed. Now with our wide choice of antibiotics we can heal our cases early. I see fewer and fewer newly developed cases of "chronic otitis externa."

I should now like to present some methods of therapy that I have found satisfactory. I do not culture all cases simply because (1) it becomes too expensive for the patient and (2) repeated cultures demonstrate the same organisms and sensitivity tests. Cultures and sensitivity tests are only resorted to where results of therapy are unsatisfactory or in old unsuccessfully treated cases. All cases of furunculosis or cellulitis are treated as follows. The canal is cleansed, and a wick saturated with polymyxin B (Aerosporin) sulfate preparation is inserted. This preparation contains polymyxin, which is most effective against Pseudomonas and Gram-negative organisms; it also contains acetic acid, which helps restore the normal pH of the canal. For home use the patient uses the following solution as drops over the wick: a chloramphenicol preparation (Chloromycetin Otic), 15 cc., and polymyxin, 25 mg. The wick is removed by the patient two days later, and the drops are continued for one week in all. I see the patient for the second time in a week, and in most instances the ear is free of all infection. The canal is cleansed and swabbed with polymyxin B solution, but if I suspect some mold, *m*-cresylacetate (Cresatin) with thymol is used. The patient is then given a prescription for 10% boric acid ointment, to be used three times weekly and before and after swimming. I selected chloramphenicol for two reasons: 1. It is stable in solution as dispensed. 2. It most frequently demonstrated its effectiveness in sensitivity testing. The addition of polymyxin enhances its effectiveness. (The further addition of

‡ References 27 and 28.

acetic acid to this mixture may be of added value?) Prior to 1955 I used a polymyxin B solution to which was added 10,000 units of bacitracin. I did not find this combination as effective.

Many early cases of external otitis present purulent drainage without pain. These cases are treated in the same manner as the cellulitis cases. The boric acid ointment is prescribed only after all evidence of infection is controlled. To prove the effectiveness of the boric acid careful, control studies were made. In bilateral cases, for example, the boric acid was used in one ear alone and recurrences took place only in the opposite ear. Where several children in a family were having recurrent attacks the boric acid was used in one member only. The patients given boric acid were rarely seen again, while those not given boric acid had recurrences. It is difficult to explain the effectiveness of boric acid. It is possible that the pH is a factor; however, a paper [30] published in 1952 demonstrated that boric acid was effective in Pseudomonas infections. By using this method of therapy I have cut down the number of repeat office visits 75%.

There is a group of cases that do not do well on the above therapy or if they are doing well on active therapy soon have recurrences. I have found many of these infections on culture to be caused by a coagulase-positive hemolytic Staphylococcus aureus. These ears usually present loose serum crusts, which when removed present a raw hyperemic oozing surface. Sometimes the ears are dry, with flaky scales. The concha and the retroauricular folds may be involved. These cases are classified as staphylococcic eczematoid dermatitis—a localized allergy to the staphylococcic exotoxin. This clinical entity has been well described by Theodore [31] in reference to the eyelids. I have found numerous patients with both lid and ear involvement at the same time. These cases are satisfactorily treated by desensitization, as described by Hansel,[32] and local use of the corticosteroids combined with neomycin.

In well-established cases of external otitis, especially of several years' duration, the therapy described is not always satisfactory. Here the problem of lowered local resistance to infection, trauma by scratching, must be handled in a different manner. A new preparation, not yet marketed at the time this was being written, has been a pleasant success. This medication, called Cortisporin otic solution, contains hydrocortisone (Hydrocortone), polymyxin, and neomycin (manufactured by Burroughs Wellcome & Company, Inc.). The hydrocortisone suppresses the itching, and the antibiotics are effective in preventing bacterial invasion. For the old chronic cases with incessant itching this preparation has been a boon. Many of these patients have found they must use this medication intermittently to maintain relief.

In my cases less than 10% are due to fungus. Clinical appearance alone suffices in most instances in making the diagnosis. The wet-paper type of debris in which are found flecks of mold is characteristic. Smears may be used to verify the diagnosis. The treatment described by Gill [8] is most effective. *m*-Cresylacetate with 1% thymol is applied locally, and for home use the solution is diluted 50% with olive oil. The ears are free of mold in about a week, and the patient is advised to use his drops intermittently to prevent and control recurrences. I have used Castellani's paint a great deal in the past but have practically discarded it because of some definite disadvantages. The patient, of course, objects to its color. The biggest disadvantage, however, is its rapid loss of potency. Unless the solution is prepared frequently it is apt to be ineffective.

REPORT OF CASES

CASE 1.—Child, aged 6, all summer long had recurrent earaches and discharge. The examination revealed bilateral green purulent discharge and marked tenderness. The patient was treated on many occasions elsewhere, but discharge and pain recurred after swimming. Treatment consisted of chloramphenicol-polymyxin ear drops for one week. This was followed by boric acid ointment. There were no recurrences after six weeks. The mother stated

that this was the first time that swimming did not bother the child's ears.

CASE 2.—Child, aged 10, for several summers had recurrent earaches following swimming. All forms of medication gave temporary relief. On each occasion the ears revealed purulent discharge and furunculosis. In the summer of 1954 the child was given chloramphenicol-polymyxin drops followed by boric acid ointment, three times weekly, before and after swimming. There have been no recurrences. For the entire summer of 1955 the child used the boric acid ointment as prescribed the previous year and there were no attacks of external otitis. It was the first summer that the child was free of ear disturbances.

CASE 3.—An adult male patient who had bilateral purulent external otitis was first seen in the summer of 1954. The patient had had a similar condition the year before. At his first office visit he was given oxytetracycline (Terramycin)-polymyxin ear drops, and in one week his ears were apparently well. One month later there was recurrence, but at this time the discharge was clear; the canals had a raw appearance. A culture taken at this time showed Pseudomonas. Patient was again given oxytetracycline-polymyxin ear drops, and the ears cleared up. I did not see the patient again during the summer of 1954, although he had several more attacks. Patient returned to me in the summer of 1955 with a fresh attack of external otitis and at this time he was given chloramphenicol-polymyxin ear solution, and after one week's period his ears were entirely cleared. There were no recurrences for the remainder of the summer.

CASE 4.—The patient, a married woman, gave a history of intermittently having ear discharge and pain all her life. The examination revealed the ear canals to be free of wax. The meatuses and canals were markedly excoriated and tender. There was a serous discharge. The culture revealed a coagulase-positive Staphylococcus aureus. She was given oxytetracycline-polymyxin ear drops, and after the ears were clear of all evidence of infection she was given neomycin-hydrocortisone ointment. Patient was started on Staphylococcus toxoid therapy. After one year there were no recurrences.

CASE 5.—An adult male patient was first seen Oct. 12, 1954, for draining and itching ears of one month's duration. Had recurrent bouts for many years. Oxytetracycline-polymyxin ear drops were prescribed, and the patient was seen one month later, at which time the ears seem to be improved but were not healed. Patient was not seen again until July 12, 1955, although he had had many repeated attacks since his last visit. At this time chloramphenicol-polymyxin was prescribed, and in two weeks the ears seemed to be improved but not healed. The patient was then given Cortisporin, and after five days there was complete cessation of symptoms. On Nov. 15, 1955, the patient had a recurrence and the ears were weeping and inflamed. The culture at this time revealed Pseudomonas. Cortisporin was prescribed again, and in three days the ears were well. Patient was advised to use the Cortisporin periodically.

CASE 6.—The patient, a married woman, presented bilateral discharging ears with itching for three months. The patient had tried all sorts of treatment and had made up her mind to have x-ray therapy. She was given chloramphenicol-polymyxin ear drops, and in one week she was asymptomatic. She was then given boric acid ointment, and when she was seen three weeks later there were no further symptoms.

CASE 7.—An adult male patient had a history of itchy ears for many years. First seen Dec. 7, 1954. Ear canals presented silvery loose scales. No wax. Concha involved. Neo-Cortef topical ointment (an ointment containing hydrocortisone acetate and neomycin sulfate) prescribed. Left ear had loose debris with clear serous drainage. The right ear was dry and scaly. Patient was seen again on Feb. 25, 1955, and was given polymyxin B with bacitracin. On March 3, 1955, there was little improvement and the ears still were itchy and wet. On March 10th the ears were better but scaling was still evident. On Nov. 25, 1955, the patient, who wore glasses, brought to my attention a condition of his eyes that had been present for several years. Both lid margins and lid surfaces presented loose scales with an underlying hyperemic surface. The lids were itchy. There was a low-grade conjunctivitis. The ears now presented cracks and fissures that wept clear serum. The retroauricular surfaces presented a similar picture. Culture of the ears revealed a coagulase-positive hemolytic Staphylococcus aureus. Staphylococcus toxoid and Cortisporin ointment for both the eyelids and the ears were prescribed. On Dec. 5, 1955, there was complete relief of symptoms and the eyelids and ears were almost normal in appearance.

CASE 8.—Patient, a married woman, was first seen in the summer of 1950 and had had itchy and draining ears intermittently for over 10 years. Patient brought with her all the drugs she had used in the past: nitrofuracin (Furacin), streptomycin sulfate, *m*-cresylacetate and thymol, Otomide,§ alcohol and salicylic acid, silver nitrate, zinc oxide and coal tar. On her initial visit the ears were painted with Castellani's paint and Neosone (an ointment containing neomycin and cortisone) was given for home use. After marked initial improvement the

§ A stable solution of 10% urea and 5% sulfanilamide in glycerin, with 3% chlorobutanol.

symptoms recurred and the patient became despondent. In 1955 patient was given Cortisporin ear solution, and after one week she informed me all symptoms had subsided. Patient has found since then that she must periodically use the solution to remain free of symptoms.

CASE 9.—Patient, a married woman, was first seen on Jan. 10, 1955, and she gave a six months' history of itchy and stopped-up ears. The right ear canal was filled with a greasy debris in which were found black sooty specks. The left ear was negative. In one week the right ear appeared well but the left ear began to itch. The left ear at this time revealed black specks, which on smear proved to be mold. Castellani's paint was prescribed for the left ear. On Feb. 1, 1955, there was a recurrence of external otitis in both ears. Examination again revealed mold. *m*-Cresylacetate with thymol at this time was prescribed, and in one week the patient was relieved of all symptoms. Patient had remained well for several months.

CASE 10.—Patient, a married woman, was first seen on June 14, 1952, with a complaint of a stopped-up left ear. Examination revealed a clear serous discharge and a wet-paper debris with black specks. Smears were positive for mold. The ears were cleansed and Asterol Tincture (2-dimethylamino-6-[β-diethylaminoethoxy]-benzothiazole dihydrochloride) was applied. Castellani's paint was prescribed for home use, and after one week the ears were clear. There have been no recurrences to date.

CASE 11.—Patient was seen in August, 1955, with a complaint of itching and running ears. This complaint had persisted all summer long. Examination of both ears revealed green purulent discharge. Chloramphenicol-polymyxin ear drops were prescribed, and in 48 hours the discharge had ceased. One week later the ears appeared well. Boric acid ointment for home use was prescribed and was also to be used before and after swimming. There were no further attacks.

CASE 12.—Patient had a history of swimming in local pool almost daily. Pain in left ear but no drainage. Examination revealed furunculosis. Treatment consisted of cleansing, swabbing with Neosporin,‖ and dusting with powder containing neomycin, oxytetracycline, and polymyxin. Wick was inserted and chloramphenicol-polymyxin drops were given. Wick removed in two days and drops continued for five more days. In one week no pain or drainage, and boric acid ointment prescribed before and after swimming. No further attacks remainder of summer in spite of constant swimming.

‖ An ointment containing polymyxin B sulfate preparation, bacitracin, and neomycin sulfate.

CASE 13.—The patient, a sister of Case 12, was in the swimming pool almost daily. Repeated bouts of furunculosis, relieved by chloramphenicol-polymyxin drops. Recurrences not controlled until boric acid ointment was used.

CASE 14.—A female patient was seen Jan. 11, 1954, with a history of itchy ears with occasional drainage for many years. Recently had a bout of furunculosis treated elsewhere. Examination revealed oozing inflamed meatuses and some scaling. Otosmosan (a liquid containing sulfathiazole urea incorporated in dehydrated glycerol) was prescribed after the ears were painted with Castellani's paint. In two weeks the ears were dry but itchiness and scaling persisted. Neosone was prescribed, and this helped temporarily. I did not see the patient again until Nov. 21, 1955. Ears were hurting, and the ear canals were swollen, oozing, and flaky. Chloramphenicol-polymyxin was prescribed. Cultures and smears demonstrated Pseudomonas and yeast. Canals wiped with *m*-cresylacetate and thymol. The chloramphenicol-polymyxin was continued for five more days. Boric acid ointment was then prescribed, but two weeks later drainage recurred. Cortisporin was given and there was immediate cessation of itching and drainage. The ears continued to remain well.

SUMMARY

A review of the recent literature regarding external otitis is given.

The seasonal incidence of external otitis for four years is demonstrated.

The "cycle of external otitis" is shown diagrammatically, and the therapeutic measures needed during each stage are given.

The successful use of chloramphenicol (Chloromycetin) and polymyxin in the "bacterial stage" of external otitis is reported.

The use of boric acid ointment in prophylaxis was effective.

Cortisporin, a preparation containing polymyxin B sulfate preparation, neomycin, and hydrocortisone, was surprisingly beneficial in "old" cases of external otitis.

Infectious eczematoid dermatitis involving the ears and, at times, the eyelids was treated with staphylococcic toxoid and antibiotic-corticosteroid combinations.

Fourteen cases are presented demonstrating the varying characteristics of external otitis and the modes of therapy.

711 Delaware Ave.

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Astrocytomas of the Nose

RAMÓN del VILLAR, M.D., México, D.F., Mexico

To the generic group of the gliomas belong the tumors designated astrocytomas, and, according to Ewing,* these neoplasias are, in general, cerebral or cerebellar tumors of slow growth, composed principally of adult glial cells. The astrocytomas have a special structure and are relatively acellular and scarcely vascularized. Microscopically, they are observed to be composed of stellar cells, between which can be seen fine cytoplasmic fibers; in some cases these fibers are scarce or nonexistent. Bailey and Cushing named them "protoplasmic astrocytomas," and, according to them, they have a greater activity than the fibrillated forms. These authors make no mention of astrocytomas other than those localized in tissue of the cerebrospinal axis.

On the other hand, localization of tumoral formations of neural tissue outside of the cerebral or cerebellar axis does occur, although rather infrequently, and the finding of such neural tissue in the nose deserves, in all cases, to be duly reported and made known.

Macomber and Wang [3] divide the congenital neoplasms of the nose into three groups, in accordance with the origin of error in development: first, tumors of neurogenic origin (gliomas, encephaloceles, and neurofibromas); second, tumors of ectodermic origin (dermoid cysts); third, tumors of mesodermic origin (hemangiomas). After a very complete study, they found, up to 1950, thirty-four cases of nasal glioma and, therefore, consider them rare congenital tumors.

In 1951, Banham and Sharp [4] described a small intranasal glioma in a child of 14 months. Cassidy and Wahl,[5] in 1954, reported two intranasal gliomas, which they described merely as tumors of anomalous tissue of the nervous system. These authors consider this type of tumor a embryonal residue of displaced neural tissue, whereas Davis,[6] who in 1942 reported a case which he called nasal fibroglial heterotropy in children, is of the opinion that neoplasms of this type belong to the group of fibrogliomatous tumors which, embryologically, originate from encephaloceles.

McGillicuddy [7] considers that nasal gliomas are congenital herniations of the cerebrum, not true neoplasms. Indeed, it is supposed that they are congenital malformations which permit the exit of normal neural tissue through one of the anterior cranial orifices, which are united to the cerebrospinal axis by means of pedicles, more or less ample and manifest, in which case the meningoceles are formed, whose formations can, therefore, be traced to fetal life and which exist as tumors of this type at birth. It may also be that in the first moments of extrauterine life the pedicle of union with the exteriorized cerebral mass of the cerebrospinal axis may disappear, leaving, thus, a tumoral mass which may be a glioma. According to Schwartz,[8] there are those of the opinion that these masses must be growths originating at the expense of neural elements exclusively of the nose.

I believe, in any case, that this latter opinion is valid for intranasal tumoral formations, which had, perhaps, intrauterine and extrauterine growth and are clearly ostensible, but

Accepted for publication Feb. 9, 1956.

Chief of E. N. T. Department, Hospital Infantil de México.

* Ewing [1] cited Bailey, Buchanan, and Bucy.[2]

not for extranasal congenital tumoral formations, whose growth is not apparent. Such is the case of the tumors of the nasal pyramid which have been observed in our service of the Hospital Infantil de México and which are the subject of this report.

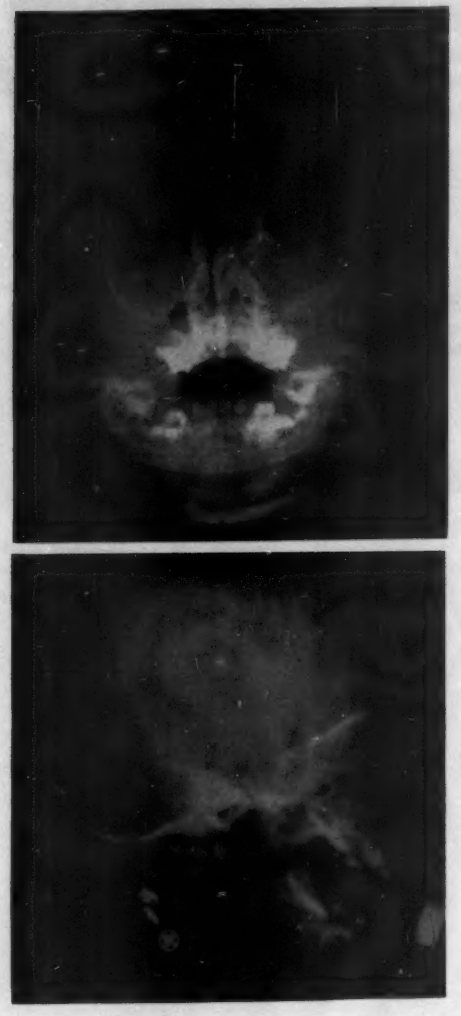


Fig. 1 (Case 1).—Above: x-ray showing the tumoral shadow. Below: lateral x-ray view.

The extranasal tumors of this type are rounded or cupular masses of firm consistency and slight hardness, of a reddish or reddish-purple coloring, easily displaceable from the deeper layers, making them, according to Hill,[9] who reports a case similar to mine, easily confused with dermoid cysts or hemangiomas. Hill insists upon the importance of differentiation between gliomas and nasal encephaloceles, because the latter may have connections with the cephalorrhachidian fluid and may be a cause of meningitis after surgical removal.

I cannot agree with Hill's opinion because, it seems to me, it is very difficult clinically to differentiate accurately a glioma from an encephalocele before surgical intervention, even though there exists the Furstenberg test (as cited by Cassidy and Wahl [5]) for determination of the possible intracranial communication of these tumors. This test includes compression of the homolateral jugular vein, which produces, when the tumor has intracranial connections, a certain tendency to expand or to pulsate.

On the other hand, in all the cases in the literature, the rarest are the types of extranasal glioma, of which I have only been able to find those mentioned by Hill,[9] Miller,[10] and Cuthbert.[11] Of the cases reported by the latter, one is probably of the mixed type, intranasal and extranasal.

As can be seen, diagnosis is difficult, so much so that in loose-leaf "Otolaryngology" Peer † asserts that a glioma can be easily confused with a dermoid cyst when it is found at the root of the nose and that differential preoperative diagnosis of dermoid cysts and nasal gliomas is not possible. It is only during the course of surgical intervention, or by means of histological examination, that the existence or absence of the pedicle may be shown.

In summary, gliomas and extranasal astrocytomas, in accordance with my own experience and the authoritative opinion of Figi and Devine,‡ are benign congenital tumors

† Peer, L. A.: Congenital Anomalies of the Nose and Sinuses, Chap. 6, p. 53, in "Otolaryngology." [12]

‡ Figi, F. A., and Devine, K. D.: Cysts and Tumors of the Nose and Accessory Sinuses, Chap. 14, p. 4, in "Otolaryngology." [12]

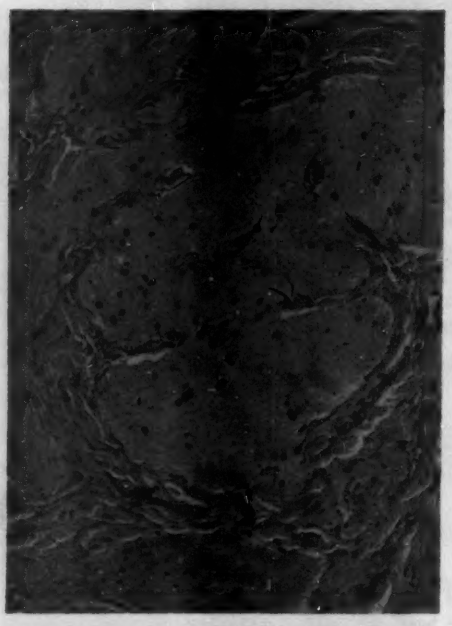


Fig. 2 (Case 1).—Photomicrographs at different magnifications showing the glial tissue; hematoxylin and eosin stain.

of a firm consistency which appear at one side of the root of the nose and which represent an anomaly of embryonal glial tissue. They have little tendency toward growth, and there exists no report of metastasis or recurrence. Indeed, those cases observed by me presented no aspects of recurrence one year postoperatively.

REPORT OF CASES

Case 1.—A 3-month-old infant was admitted on Oct. 13, 1954, to the Ophthalmological Service of the Hospital Infantil, from Jejutla, Oax., with history of a "lump" of slow growth, on the internal angle of the left eye, level with the nose, from birth. There was constant tearing and an abnormal secretion from this eye. Upon general examination the patient was found to be normal except for the tumor noted. E. N. T. examination, made by me, revealed the following: In the left ethmoidal region, immediately below the orifice of the nasolacrymal passage, there existed a tumorous mass the size of a small hazelnut, spheroid in shape, with a hardness similar to that of a fibroma or chondroma, wine-red in color, over which the adjacent structures were easily movable, the tumor itself being semimovable over the deeper structures; its location, more or less, was noted at the suture of the rama ascendentis supramaxillaris and the os nasalis, there being no apparent separation of the suture on taction. Radiologic diagnosis: "Osteoma, with malformation of the left frontal sinus, which is amply communicated with the right frontal sinus, forming a large cavity over the root of the nose, and giving the impression

Fig. 3 (Case 2).—*A* and *B*, two views of the tumor "in situ" before operation. *C*, view five days after excision of the tumor.

of a possible frontal meningocele. The lateral plate shows dehiscence of the nasofrontal region. The image is compatible with that of a Meningocele." My interpretation of the radiological studies led me to affirm the following: First, no frontal sinuses existed, as was to be expected, considering the patient's age. Second, there was no osteomal shadow in the region ordinarily occupied by the sinuses. There was a tumorous shadow, implanted by means of a wide base, directly over the bone in the aforementioned region, without apparent penetration of the same. Third, the tumorous shadow corresponded with a nonosseous neoplasm. Fourth, the tumorous shadow extended downward and inward, invading the maxilloethmoidal zone of the left maxillary antrum (Fig. 1). Clinical diagnosis: I thought that this was a congenital tumor of a fibrous or chondromatous nature, implanted on the external wall of the nose, with probable true origin in the anterior ethmoid.

As can be seen from the above transcription of the patient's records, we did not use Furstenberg's test for the differential diagnosis, nor did we, at any time, consider the possibility of an extrinsic neurogenic tumor of the nose. Under these conditions, we proceeded to surgical removal of the tumor.

Surgical intervention: An incision, 2 cm. in length, was made from the root of the nose to the alar margin following the nasolacrymal sulcus, including the periosteum. The lacrymal sac was separated and protected out of the field. The tumor found was of a fibrous nature, oval-shaped, encapsulated, about the size of a hazelnut, with a pedicle inserted into an osseous aperture existing in the suture of the rama ascendentis supramaxillaris

and the os nasalis, which was implanted in the anterior ethmoidal cells and which did not appear to continue inward or upward. A blunt dissection was performed, without causing perforation of the nasal mucosa or opening of the nasal cavity. The cutaneous wound was closed with individual non-absorbable surgical (silk) sutures. The operative

Fig. 4 (Case 2).—Above: x-ray showing the tumoral shadow. Below: lateral x-ray view.

diagnosis was that of "dermoid cyst." The specimen was sent to the department of histopathology, Dr. Maximiliano Salas in charge, for study. The report was as follows:

Histopathological study, macroscopic description: The specimen was that of a tissue fragment of oval and regular shape, measuring 2×1.4×1 cm. at its greater diameter. It was of smooth surface and hard consistency and appeared to be almost entirely covered by mucosa. Microscopic description: In preparations stained with hematoxylin and eosin it was observed that the fragment was formed almost exclusively of glial tissue, whose cells were astrocytes. The tissue was disposed into regular layers, which were divided by bands of connective tissue, where some blood vessels could be seen. Around the glial tissue, both muscular and dense connective tissue could be observed, as well as a small amount of adipose tissue. Diagnosis: glioma (astrocytoma) of the root of the nose (Dr. O. Angulo), signed: Dr. Max Salas (Fig. 2).

Case 2.—A 25-day-old infant was admitted on Oct. 21, 1954. Ailment: mamelonated tumor, about 2 cm. in diameter, from birth, ligated at the base and excised 48 hours after birth. Two days later there was a local hemorrhage and ligation and fulguration were practiced. Since then it continued to increase in size and volume until it measured 1 cm. in diameter. General examination: Upon admission to Ward I, Surgery, at the Hospital Infantil, the patient weighed 3,400 gm., had a temperature of 36.4 C (97.5 F), and was conscious, well nourished, and well hydrated; the fontanelle was under normal tension, and the eyes and ears were normal. A sessile tumor, about 1 cm. in diameter, was present at the root of the nose, between the eyebrows, wine-red in color, with signs of hemorrhage and ligation; it was movable and semihard. Circulatory system, normal. Respiratory tract, normal. Abdomen: soft, pliable; umbilical orifice about 1 cm. in diameter. Phimosis. Extremities normal. Nervous system, normal. Ganglionic system, normal.

E. N. T. examination, made by me, revealed a mamelonated tumor at the aforementioned site, apparently derived from the skin of the ciliar region in its internal portion, movable over the adjacent layers, rounded at its base, about 1.5 cm. in diameter. Its most prominent portion presented a mummified area of reddish-black coloration, below which could be seen a ligature (Fig. 3). Around the base was noted a depression, as though there might exist dehiscence of the ethmoid. There were no abnormalities in the interior of the nasal fossae. Furstenberg's test was negative. Clinical diagnosis: probable extrinsic nasal neurogenic tumor, not united to the cerebrospinal axis. Complementary laboratory examinations revealed normal biometry and negative Kahn, Wassermann, and Mazzini tests. Radiological interpretation: The cranial cavity presented no dehiscence compatible with frontal meningocele; however, the ethmoid region, in the lateral plate, showed separation of the osseous planes, giving the impression of a tumor of sinusal origin or of a

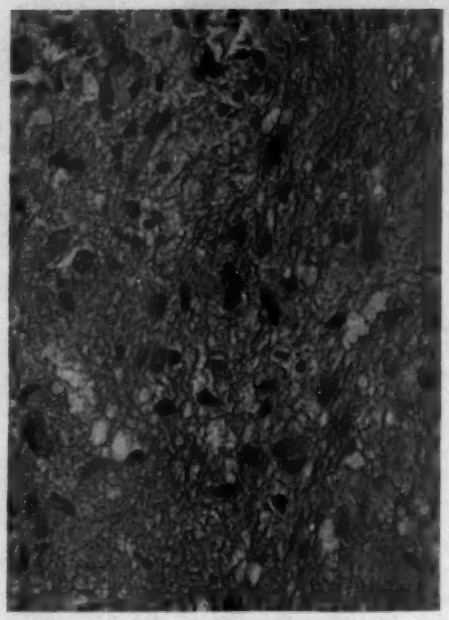


Fig. 5 (Case 2).—Photomicrographs at different magnifications showing the glial tissue.

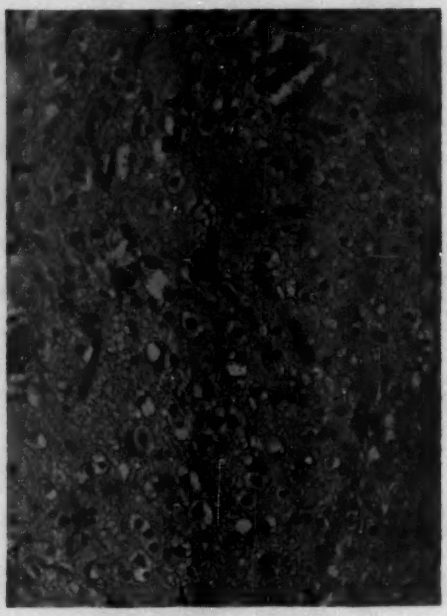
mucous-like process, much like the mucoceles of that region, which may be covered with normal skin (Fig. 4).

It will be noted that in this case, similar to the previous case, we considered from the beginning the possibility of a nasal glioma and made a differential diagnosis, using Furstenberg's test, which was negative, to determine whether or not the neural tissue was united to the cerebrospinal principal axis. In view of the results, it was decided to practice surgical removal.

Surgical extirpation: A vertical, spindle-shaped incision of the skin was made, which included the granuloma, under which, though not adhered to it, was found the principal tumorous mass. Apparently, this mass did not communicate with the cranium and was fixed, by a thin pedicle, through a small aperture in the suture of the os nasalis and the maxillary process, to a small ethmoidal cell. The specimen was sent to the histopathology laboratory for study.

The following is the laboratory report on the specimen:

Macroscopic description: The specimen consisted of a small irregular-shaped tumorous mass, approximately 2.2×1.5 cm. at its greatest diameter, covered with mucosa at different points; its consistency was slightly increased. Microscopic description: Preparations stained with hematoxylin and eosin showed tissue formed by cells whose nuclei were large and vesiculated, with apparent nucleoli. The tumor had few cells, though intercellular acidophile tissue was abundant. These elements seemed to correspond to glia (astrocytes). The cells were disposed into small and irregular layers surrounded by dense connective tissue, around which



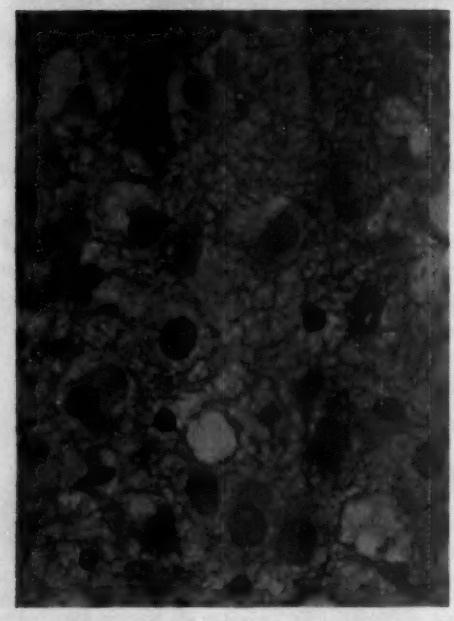


Fig. 6 (Case 3).—Photomicrographs at different magnifications showing the glial tissue.

could be seen muscle tissue, adipose tissue, and a few blood vessels. Histological diagnosis: probable nasal glioma (astrocytoma) (Dr. O. Angulo), signed: Dr. Max Salas (Fig. 5).

It was my good fortune to learn of another case, similar to the others, which had not been properly recorded in the clinical archives of the hospital because both diagnoses, at admittance and upon discharge, were dermoid cyst of the nose and, also, because the patient was not sent to the E. N. T. services but, rather, to General Surgery. The following is the case history:

Case 3.—A one-month-old baby girl was admitted to Surgical Service III, June 7, 1953. Diagnosis on admission: probable cyst of the nose. Family history: tuberculosis and syphilis negative in the mother, unknown in the father. Personal history: first child, born at term, of normal childbirth, received regular maternal feeding. Ailment: A tumor was noted, from birth, at the right side of the nose, near the root, which had no apparent increase. Physical examination: conscious suckling infant, afebrile, well nourished, and with a tumor, about the size of a bean, adherent to the skin and of a hard consistency, present at the root of the nose, on the right side. The rest of the physical examination was negative. Laboratory examination: Hematic biometry showed moderate leucocytosis. No radiologic studies were made. Evolution: With the patient admitted under the conditions mentioned above, it was decided to operate three days after admittance, at which time, under general endotracheal anesthesia, without any complications, Dr. Manlio Hernández

performed the extirpation of the cyst. Five days later, June 16, 1953, the patient was discharged with a final diagnosis of dermoid cyst of the nose.

However, routine histopathological examination revealed the following: Macroscopic description: The specimen consisted of a small tumorous mass, of irregular shape, which measured 2×1.6×1 cm. at its greater diameter. The exterior surface was irregular, of a pale-red color and diminished consistency. The specimen was placed in 10% formaldehyde solution for fixation and future study. Microscopic description: The tumor had a minimal amount of fibrous and adipose tissue at its periphery, with some smooth-muscle fibers at one of its extremities. The greater part was composed of glial tissue, well vascularized. The smaller blood vessels had a limited amount of collagenous tissue, forming thin partitions which divided the tumor. Staining with hematoxylin and eosin showed what seemed to be nerve fibers. In some of the partitions the fibrous tissue was composed of astrocytes. Diagnosis: nasal glioma (astrocytoma) (Dr. O. Angulo), signed: Dr. Max Salas (Fig. 6).

From the cases presented, it can be seen that a glioma (astrocytoma) of the external wall of the nasal pyramid is a tumor of a congenital nature and of embryonal origin. Not neoplastic, it can be considered a true encephalomeningocele if the pedicle uniting the herniation to the neural axis during intrauterine life persists after birth, but if the pedicle is broken and totally absorbed, or if it is only vestigial, the isolated tumorous mass is a glioma.

Experience shows, also, that it is very difficult, owing to the extranasal diversity of the glioma, to make an exact previous differential diagnosis. Even Furstenberg's test, so valid for the intranasal variety, is practically worthless for the extrinsic types. Besides, if Furstenberg's test should be negative, doubt would still remain as to whether there is a tumor of neural tissue or a dermoid cyst. Where the osseous orifice is quite extensive and the pedicle proportionally large, x-rays can give a differential diagnosis between glioma and dermoid cyst.

In conclusion, I repeat that since the extranasal gliomas are not neoplasms they behave like benign tumors, and there is no reason that they should give way to metastasis or recur, except, perhaps, as a result of incomplete surgical removal.

The Department of Pathology of the Hospital Infantil and, in particular, Dr. Max Salas, Head of the Department, and Dr. Octavio Angulo gave excellent reports and valuable opinions. Prof. Isaac Costero, M.D., Head of the Department of Pathology of the Instituto Nacional de Cardiología, gave his counsel. Collaboration and excellent work was performed by Mr. Sivilla, of the Department of Photography of the Hospital Infantil.

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Laryngoesophagectomy

Primary Closure with Laryngotracheal Autograft

MAX L. SOM, M.D., New York

Carcinoma of the postcricoid region involving the mouth of the esophagus and the posterior surface of the larynx is best treated by surgical extirpation. Lateral pharyngotomy, as suggested by Trotter,[1] with preservation of the larynx is a limited operation with disappointing results and has been abandoned. Radical excision of a segment of the pharynx and the upper portion of the esophagus, together with the whole of the larynx, has more recently been performed. The alternative is the relegation of the patient to a painful and lingering death.

Reconstruction of the pharynx after a pharyngoesophagolaryngectomy has heretofore required prolonged hospitalization and extensive plastic repair. The operation suggested by Wookey[2] provided the posterior surface of the new esophagus but bipedicle skin flaps were almost invariably required for closure. In many instances a prolonged pharyngostomy, with all its discomforts, was required, and often recurrence of the carcinoma appeared before the final stage of the plastic closure. If preliminary skin flaps were raised to effect an earlier reconstruction of the pharynx, surgical eradication of the cancer had to be postponed and the chances of success or even palliation were minimized.

Primary reconstruction of the continuity of the pharynx and esophagus after pharyngoesophagolaryngectomy was first performed by V. E. Negus[3] and Rob,[4] in 1949. The procedure consisted in the introduction of a large plastic tube into the cut ends of the pharynx and esophagus and encircling the tube with a split skin graft. The graft was sutured around the tube so as to completely encircle it.

In 1952, Edgerton[5] reported a one-stage reconstruction of the pharyngoesophagus in which he employed a split skin graft over a cone of tantalum mesh fashioned to replace the segment of excised esophagus. In December, 1953, Dr. John J. Conley[6] described a procedure illustrating a one-stage radical resection of the cervical esophagus, larynx, and pharynx with immediate reconstruction. The created mucosal deficiency was corrected by an end-to-end anastomosis of a free split-thickness graft to the pharyngeal and esophageal openings. Wire-mesh and plastic stents were fashioned to support the grafts and maintain an esophageal lumen. Stenosis was the most unpleasant complication.

The obvious advantage of primary reconstruction is the completion of the surgery in one stage, with avoidance of the unpleasantries of a pharyngostomy. The plastic tube is allowed to remain in situ about six months. The patient swallows by the normal route after a relatively short interval. The development of strictures at the proximal and distal anastomoses is a constant source of trouble.

In 1954, Asherson[7] reported a case of an early carcinoma of the hypopharynx, limited to the posterior wall, in which a segmental pharyngoesophagectomy was performed. The larynx was separated from the esophagus by splitting the party wall and peeling the anterior esophageal mucosa from the cricoid. The larynx was then used as a tube to reconstruct the continuity of the pharyngo-

Accepted for publication March 26, 1956.

From the Head and Neck Group of the Surgical Service of the Montefiore Hospital.

esophagus. A permanent tracheostoma was created below the line of resection. Such preservation of the entire larynx is rarely feasible in hypopharyngeal lesions, since the cancer almost invariably infiltrates the cricoid and does not permit a wide excision without sacrifice of the larynx. In isolated instances, when such limited excisions are possible, the Trotter operation seems indicated, with preservation of the normal laryngeal function and use of a free skin graft to replace the excised segment of esophagus.

In November, 1955, Wilkins [8] described an operation for immediate reconstruction of the cervical esophagus somewhat similar to that presented by Asherson. His procedure differed in that he removed most of the cartilaginous framework of the larynx and trachea and preserved their mucosal surfaces to replace the excised segment of esophagus. The cricoid cartilage, which he considered a barrier to spread of the neoplasm anteriorly, was removed in continuity with the tumor after the laryngeal mucosa had been peeled away. The membranous party wall between the upper trachea and esophagus was split, and the tracheal mucosa was freed up to the cricoid. Except in rare instances, where the neoplasm is strictly confined posteriorly, such limited resection of soft tissues would appear inadequate.

Whereas in most instances of carcinoma of the hypopharynx the cricoid and the posterior wall of the larynx are involved, the anterior portion of the larynx is nevertheless free of disease. It thus would seem possible to preserve the anterior half of the larynx in the great majority of cases. Similarly, the anterior circumferences of the cricoid and the upper tracheal rings could be safely retained after wide excision of the malignant tumor in continuity with the posterior portion. These semicircular cartilaginous structures and the intervening mucosa and mucoperichondrium could be preserved to form the anterior half of the newly constructed esophagus. The posterior half could be supplied by a split skin graft resting on the prevertebral fascia and sutured to the margins of the laryngeal autografts laterally. This was in fact done and is now being reported. A hypopharyngeal carcinoma involving the entire circumference of the postcricoid region was excised in continuity with the posterior portion of the larynx and upper trachea. The preserved anterior structures were used as an autograft to form the front part of the new esophagus.

REPORT OF A CASE

A 60-year-old woman was referred to the Montefiore Hospital on Aug. 19, 1955, with a six-month history of progressive dysphagia. At the time of admission, there was inability to swallow anything but fluids. Blood study revealed the following: red blood cells, 3,520,000; hemoglobin content, 10.9 gm/100 cc.; white blood cells, 4750; polymorphonuclears, 55%; lymphocytes, 44%; stab cells, 1%.

Mirror examination of the larynx showed no abnormality. There was no evidence of neoplasm in the hypopharynx, and the motility of the vocal cords was unaffected. No cervical lymph nodes could be palpated. A profile x-ray of the pharynx showed retention of the barium swallow. Roentgen examination of the chest was normal.

Fig. 1.—Drawing of the stenosing lesion in the postcricoid region. The dotted line illustrates the line of excision of the tumor and the surrounding tissue to be sacrificed. A tracheostomy is performed below the completely divided trachea.

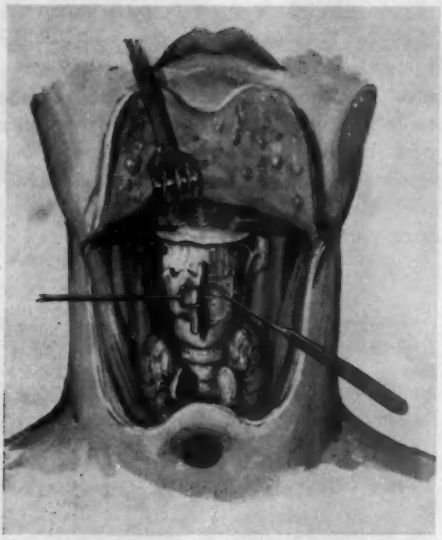
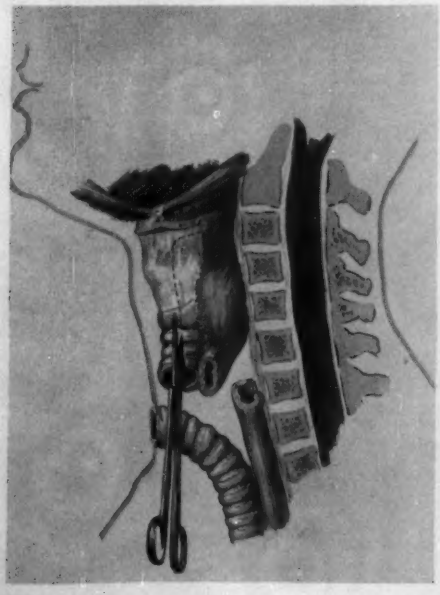


Fig. 2.—The apron incision is illustrated with the tracheostoma in the suprasternal region. The pretracheal muscles have been excised, and the thyroid isthmus is doubly ligated. The thyroid cartilage is separated from the soft tissues of the larynx, care being exercised to preserve the superior laryngeal vessels.

Fig. 3.—The trachea and esophagus have been diagrammatically transfixed below the distal level of the neoplasm. The broken line indicates the line of excision upward through the trachea, cricoid cartilage, vocal cords, and thyrohyoid membrane into the hypopharynx.



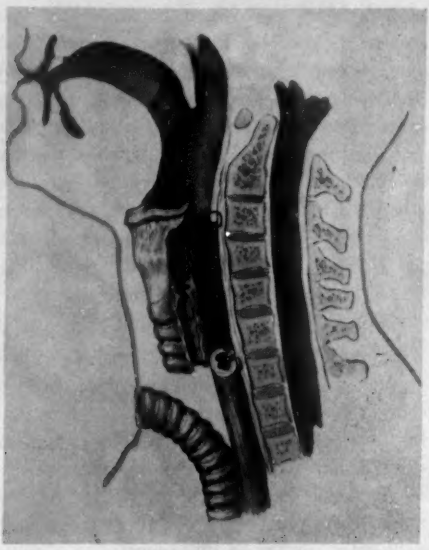


Fig. 4.—Drawing shows the defect created by excision of the neoplasm. The preserved anterior portions of the trachea and the larynx are seen suspended from the hyoid bone.

Hypopharyngoscopy revealed a stenosing lesion at the level of the cricopharyngeus muscle. The surrounding mucosal surface was thickened and nodular. A biopsy specimen was taken, after which a No. 12 Jackson bougie was introduced into the upper esophagus. A narrow-lumen esophagoscope was now passed over the bougie. It was determined that the lesion involved a segment of about 2.5-3 cm. in length. A plastic feeding tube was inserted

Fig. 5.—Drawing illustrating the anastomosis of the distal portion of the laryngotracheal autograft to the anterior circumference of the esophagus. Insert shows the Negus type of hollow tube used as a temporary mold.

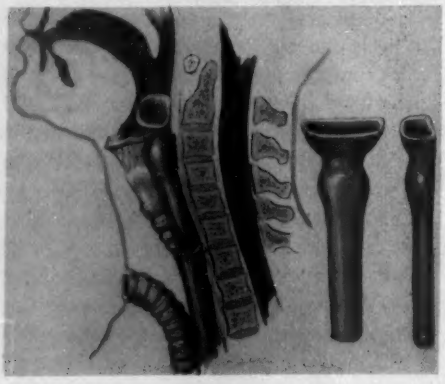


Fig. 6.—Drawing indicating the completed operation. The new segment of the pharyngoesophagus, encircling the mold, is formed by the laryngotracheal autograft anteriorly and a free skin graft posteriorly. A nasogastric tube is passed through the mold.

into the stomach, through the esophagoscope, for alimentation. The biopsy report was squamous-cell carcinoma.

Operation (Aug. 23, 1955): Local anesthesia was employed until a tracheotomy was performed, after which the patient was maintained on controlled respiration with the use of thiopental (Pentothal) sodium.

A U-shaped incision was made from one mastoid tip to the other, leaving a tongue of skin above the suprasternal notch through which the tracheostoma was to be made. The pretracheal muscles were severed above and below, and the thyroid isthmus was transected in its midportion and separated from the trachea laterally. The thyroid alae were skeletonized and their borders freed of their musculature attachments. The upper cornua were delivered, and the lower cornua were severed from the cricothyroid joint. The entire thyroid cartilage was then separated from the underlying soft tissue by sharp and blunt dissectors. Care was taken not to disturb either the superior laryngeal vessels or the inferior thyroid arteries. The trachea was transfixed between the fourth and fifth tracheal rings, and a permanent tracheostoma was created through the tongue of skin below.

By palpation the neoplasm in the postcricoid region was outlined, and the esophagus was then severed about 1.5-2 cm. below the distal tumor margin. The lower end of the esophagus was anchored to the prevertebral fascia by two nonabsorbable surgical (silk) sutures to prevent retraction. The proximal portion of the trachea was examined from below and appeared free of disease. The trachea was now bisected into anterior and posterior halves by two vertical incisions through the midpoint of its lateral walls. The incision cut through the cartilage of the trachea and continued upward through the cricoid. The anterior portion, containing the arch of the tracheal cartilages and the ring portion of the cricoid, was preserved. The posterior half, including the party tracheoesophageal wall and the wide portion of the cricoid cartilage and arytenoids, was left attached to the tumor mass for excision in continuity.

Fig. 7.—Surgical specimen showing the excised parts: *A*, the projecting portion of the epiglottis; *B*, the inner aspect of the thyroid alae; *C*, postcricoid carcinoma (horizontally placed). The arytenoids can be identified on the left. The tumor extends from above 4.5 cm. up to 7.5 cm., and then the mucosa of the cervical esophagus is seen.

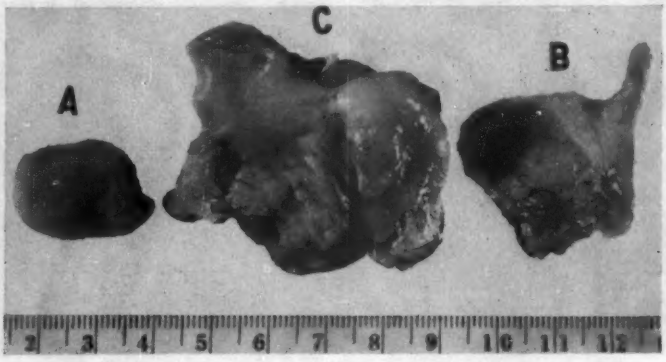


Fig. 8.—A profile pharyngoesophagogram taken one week postoperatively. The air column in the hollow mold outlines the lumen of the new esophagus. Portions of the tracheal cartilages are visible in the anterior-inferior wall, which is formed by the tracheal autograft.

The vertical incision was extended upward through the thyrohyoid membrane, and the pyriform fossa was entered. The upward extent of the neoplasm could now be easily visualized and examined. A transverse incision was now made in the pharynx about 2 cm. proximal to the upward extent of the tumor. The pharyngeal mucosa was tethered by sutures to the prevertebral fascia. The tumor, now encompassed, was easily separated from the prevertebral fascia and delivered en masse. The specimen contained the unopened resected segment of the esophagus with the contiguous posterior portion of trachea and party wall as well as the posterior half of the larynx.

The preserved portions of the larynx and trachea remained suspended by the thyrohyoid membrane

Fig. 9.—Roentgenogram, taken six weeks postoperatively, shows the barium outlining the new pharyngoesophagus.

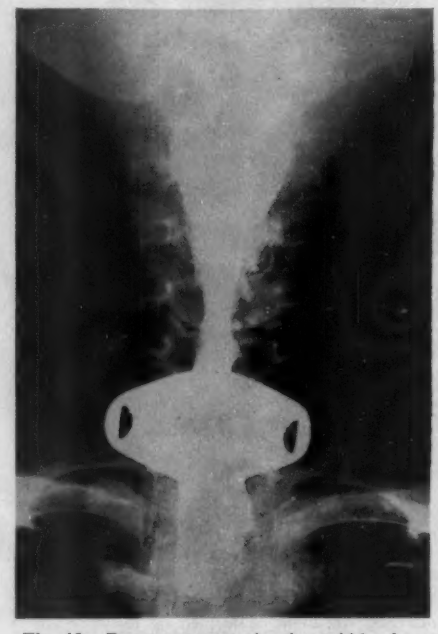


Fig. 10.—Roentgenogram showing width of new pharyngoesophagus at the proximal anastomosis.

Fig. 11.—Photograph of patient seven months postoperatively.

and glossoepiglottic and pharyngoepiglottic ligaments. The line of excision through the trachea and cricoid had cut through the midportion of the vocal cords, which retracted anteriorly. The epiglottis was amputated at its base. The mucosal surfaces were approximated by fine absorbable surgical (gut) sutures. The autograft was now swung slightly posteriorly and sutured to the anterior circumference of the lower portion of the transected esophagus. Interrupted everting 0000 chromic absorbable surgical sutures were used. A flattened hollow latex rubber tube of the type used by Dr. V. E. Negus was introduced along the anterior wall of the newly created pharyngoesophagus. The narrowed terminal portion of the flexible mold was inserted into the lumen of the distal end of the esophagus. The proximal widened orifice was first compressed and then slipped into the pharynx. The mold is so constructed that the expanded pharyngeal portion gradually narrows for a short distance and then widens again. A collar is thus fashioned at the level of the base of the tongue. This seems to hold the mold in place and prevents both regurgitation of the mold into the pharynx and downward displacement. The mold is used to bridge the gap extending from the base of the tongue to the transected esophagus. The distal, tapered end of the mold must be easily accommodated in the esophageal lumen to prevent necrosis.

A Thiersch skin graft, measuring 4 by 7 cm., was taken from the outer aspect of the thigh and spread out on the prevertebral fascia between the transected pharyngeal and esophageal mucosa. The skin surface was wrapped around the rubber mold with the raw surface toward the vertebral fascia. The graft was first sutured to the pharyngeal mucosa above and then to the posterior surface of the esophageal mucosa below. The skin graft was then sutured to either side of the laryngotracheal autograft, so that a complete mucosal and skin surface encompassed the mold and formed the new pharyngoesophagus.

The lobes of the thyroid gland are draped about the exposed surface of the suture line laterally. The nourishment of the free skin graft is derived from the prevertebral fascia, to which it is anchored. A Levin feeding tube was passed into the stomach through the rubber mold. The tracheostoma was completed, and the skin was closed after drains were placed laterally. The carotid sheaths were exposed bilaterally, and the internal jugular veins were stripped of their overlying lymph-bearing tissues, but no metastatic nodes were encountered. An elective neck dissection was not performed, since it could not be determined which was the side of maximal growth.

The after-treatment was similar to that of any laryngectomy. There was no leakage of saliva and, consequently, little discomfort. A mild infection of the left lower angle of the wound developed but promptly subsided with counter drainage.

On the 16th postoperative day, the patient was able to swallow fluids without a leak. Two days later the nasal feeding tube was removed, and on the 23rd day the rubber mold was extracted through the pharynx. The wound was completely healed one month after surgery, and the patient has continued to swallow well to date (eight months postoperatively) without any clinical or roentgen evidence of stricture formation.

SUMMARY

A case of carcinoma of the postcricoid region involving the entire circumference of the mouth of the esophagus is described. An operation for the excision of the neoplasm in continuity with the posterior half of the larynx and the party wall of the upper trachea is described. After removal of the thyroid cartilage, the remaining anterior portion of the larynx and trachea is used as an autograft to form the anterior wall of the new pharyngoesophagus. Care is exercised to preserve the superior laryngeal vessels. A Negus-type latex rubber mold is inserted, and a Thiersch skin graft is wrapped around the posterior aspect of the mold, so that the mold is completely encompassed by the mucosal lining of the larynx and trachea anteriorly and the skin graft posteriorly. A nasal feeding tube is introduced for alimentation.

In the case reported the discomfort of drooling saliva accompanying temporary pharyngostomy has been avoided and thus far strictures have not developed. The laryngotracheal autograft remained viable and supplied the anterior wall of the pharyngoesophagus. The entire hospital stay was relatively short. The presence of mobile metastatic lymph nodes is no contraindication, and it is suggested that block dissection be carried out on the side of maximum growth. The value of this procedure should now be judged by the palliation produced and this in turn by the ability of the patient to swallow after surgery. Critical analysis as to ultimate cure of primary reconstruction must be postponed, since the reported cases are as yet too few and too recent.

121 E. 60th St.

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News and Comment

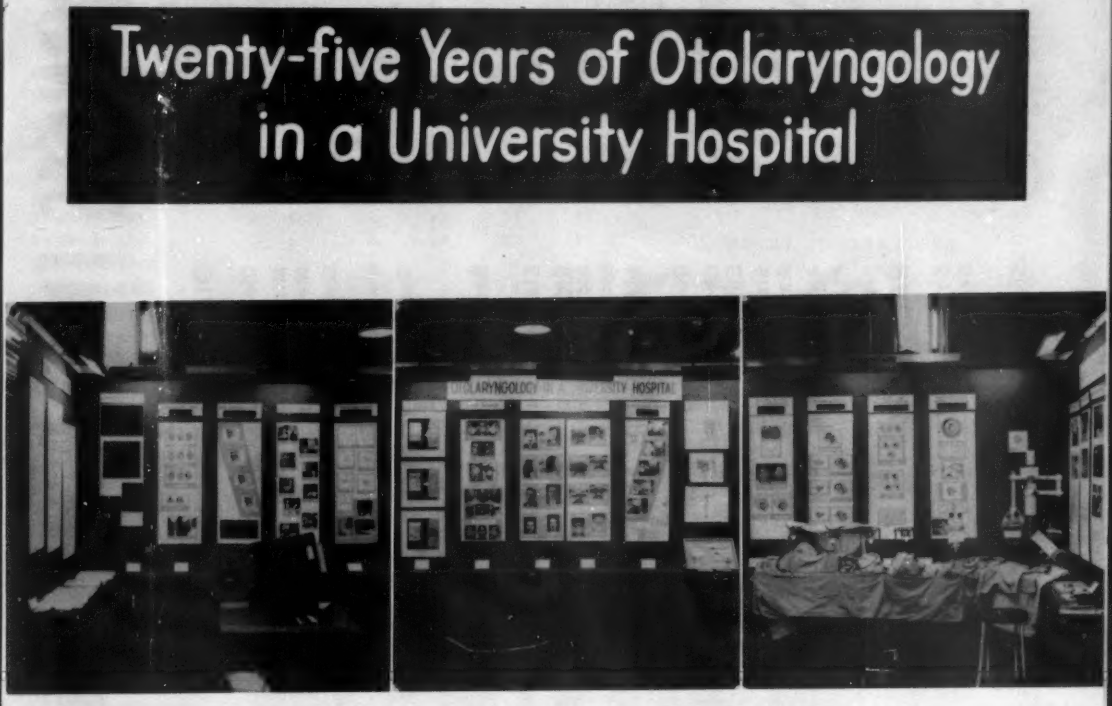
ANNOUNCEMENTS

Meeting of the Pennsylvania Academy of Ophthalmology and Otolaryngology.—The 1956 meeting of the Pennsylvania Academy of Ophthalmology and Otolaryngology will be held at the Bedford Springs Hotel, Bedford, Pa., from May 24 to May 27, 1956.

Scientific Exhibits

TWENTY-FIVE YEARS OF OTOLARYNGOLOGY IN A UNIVERSITY HOSPITAL

EDMUND P. FOWLER Jr., M.D.; FRANZ ALTMANN, M.D.; D. C. BAKER Jr., M.D.;
V. EPANCHIN, M.D.; H. S. FRIEDMAN, M.D.; BELA MARQUIT, M.D.,
and DONALD MARKLE, M.A.
New York



This exhibit was designed primarily to indicate the scope of modern otolaryngology. The emphasis is on the contributions to the general subject which have been made in our hospital. It is often said, because the number of simple mastoidectomies has decreased markedly since the advent of antibiotics, the specialty in general has contracted. That this is erroneous is brought out by statistics on page 3. Here it is apparent that simple mastoidectomy even in its heyday constituted only about 5% of operations of the ear, nose, and throat. Furthermore, with three less beds and eight less attending surgeons this hospital has more than doubled the number of operative cases in the last 20 years. Antibiotics have helped, for they permit rapid turnover of patients. The patients can now more easily tolerate the procedures that are shown in the several panels. Previously much such surgery was virtually impossible because of secondary infections.

Otolaryngology is a multifaceted specialty that changes with each new advance in medicine, just as do the other specialties. These different aspects are shown on the following pages.

From Columbia-Presbyterian Medical Center.

Shown as a scientific exhibit of the Section on Laryngology, Otology and Rhinology, at the 104th Annual Meeting of the American Medical Association, Atlantic City, June 6-10, 1955.

ADVANCES IN EAR, NOSE AND THROAT

FUNDAMENTAL EAR, NOSE AND THROAT-1930

DIAGNOSTIC METHODS

History
Observation with specula and mirrors
Palpation
Transillumination
Irrigation
Odor and color of exudates
Tuning forks and voice
Hand rotation and mass colorics
General - Temp., Urinalysis, Blood count, Lumbar puncture, Biopsies

MEDICAL TREATMENT

Analgesic	Antiseptics
Heat and cold	Dehydration
Shrinkage	Irrigation
Steam	Argyrol
Astringents	Toxin-antitoxin

SURGICAL TREATMENT

Incision & drainage *(including Sinus & Mastoid Surgery)*
Tracheostomy for life saving
Tonsillectomy and adenoidectomy
Laryngofissure
Foreign body endoscopy
Blind bougineage
Laryngectomy
Septum surgery, Cosmetic surgery
Jugular vein ligation

RADIOTHERAPY for CANCER

Primitive

RESEARCH

Clinical

REHABILITATION

Lip-reading, Carbon hearing aids, Speaking tubes, "Lost Cords League", Artificial Larynx, Social Service

TEACHING by LECTURES

Exclusively

BY 1955

DIAGNOSTIC METHODS

Diagnostic endoscopy
Minimal stimulus vestibular (balance) tests
Calibrated hearing tests
Special roentgenography
Bacteriology and sensitivity tests
Electronic tests for facial paresis
General: Nasopharyngoscope, Papanicolaou smears, psychodiagnostic techniques, constitutional studies, etc.

MEDICAL TREATMENT

Cold steam, detergents, oxygen and helium
Chemotherapy
Enzymes
Steroids
Antibiotics
Anti-histamines
Circulation therapy
Prisms

RADIOTHERAPY

Superficial
Deep
Supervoltage

BY 1955

SURGICAL TREATMENT

Airway for laryngeal paralysis
Enblock dissection
Hemilaryngectomy
Simple, modified, and radical mastoidectomy, tympanoplasty
Fenestration, stapes mobilization
Facial nerve decompression and graft
Labyrinth destruction
Skin grafts
Rhinoplasty
Otoplasty
Surgery with radiotherapy
Tracheostomy

Speech training
Fitting of hearing aids
Esophageal voice

TEACHING

Medical students
Residents
Endoscopic treatment

REHABILITATION

Psychotherapy
Auditory

RESEARCH

Laboratory
Clinical

CHANGING STATISTICS

DISTRIBUTION OF COMMON TYPES OF EAR, NOSE AND THROAT CASES AT FIVE YEAR INTERVALS FOR TWENTY YEARS.

	1933	1938	1943	1948	1953
T & A's	1021	1376	1067	1456	1308
ENDOSCOPY	170	279	201	598	738
SMR OF NASAL SEPTUM		148	165	257	144
SIMPLE MASTOID SURGERY	88	102	17	21	13
RADICAL MASTOID SURGERY	33	63	87	64	70
RADICAL SINUS		28	16	27	72
SIMPLE SINUS		89	59	89	88
RHINOPLASTY		33	27	78	72
TRACHEOTOMY	8	19	17	18	43
FENESTRATION	0	0	10	27	26
LARYNGOFISSURE		4	0	5	7
LARYNGECTOMY		2	5	9	10
ARYTENOIDECTOMY		0	0	7	3
RADICAL NECK DISSECTION		0	0	1	43
EPISTAXIS		5	21	41	33
MENIERE'S DISEASE		0	12	33	17

TOTAL ADMISSIONS TO SIXTY BEDS

1933	1938	1943	1948	1953
1787	2773	2405	3001	3292

TOTAL OPERATIONS

1598	2551	2109	2550	3046

MEDICAL EAR, NOSE AND THROAT.
1954

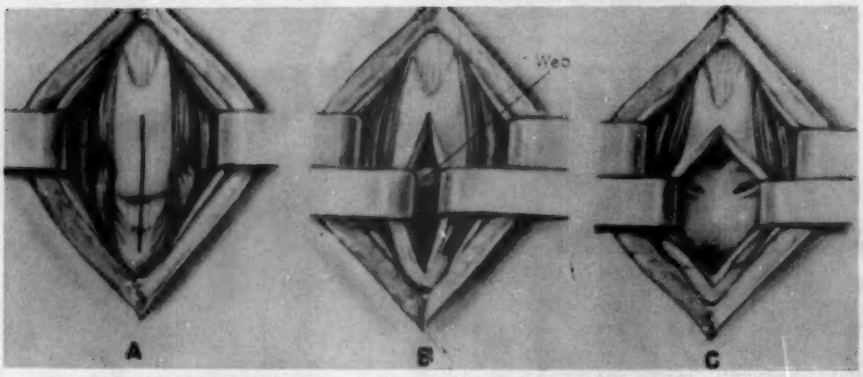
Conditions listed only if more than five patients with a given disease were admitted to the Service.

	WARD	PRIVATE
ACUTE PHARYNGITIS	12	0
ASTHMA	2	4
BILATERAL ABDUCTOR PARALYSIS	3	11
BRONCHIAL STENOSIS	15	3
CHRONIC BRONCHITIS	16	47
CHRONIC LARYNGITIS	17	16
CONGENITAL LARYNGEAL STRIDOR	2	3
EPISTAXIS	45	16
FACIAL PARALYSIS	3	2
FURUNCLE OF THE NOSTRIL	5	0
HIATUS HERNIA	4	11
LABYRINTHOSIS	2	3
LEUKOPLAKIA OF VOCAL CORD	3	2
MENIERE'S DISEASE	17	23
MUCOCOELE OF FRONTAL SINUS	3	4
PERITONSILLAR ABSCESS	37	0
PANSINUSITIS	9	9
SINUSITIS, ETHMOID	2	19
SINUSITIS, FRONTAL	6	6
SINUSITIS, MAXILLARY	36	37
SINUSITIS, SPHENOID	0	6
STRICTURE OF ESOPHAGUS	3	2
ULCER OF ESOPHAGUS	2	6

REPRESENTATIVE OPERATIONS 1954

Listed as ward or private. 95% ward surgery done by residents.

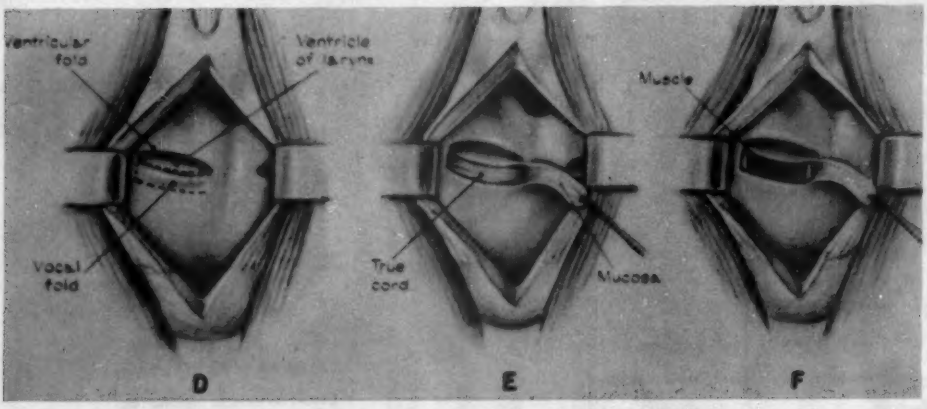
	WARD	PRIVATE
ADENOIDECTOMY	0	8
CALDWELL-LUC	27	24
CLOSURE OF ORO-ANTRAL FISTULA	5	4
FACIAL NERVE DECOMPRESSION	2	2
FENESTRATION	4	11
INTRANASAL ANTROTOMY	10	17
INTRANASAL ETHMOIDECTOMY	4	20
LABYRINTHOTOMY (DAY OPERATION)	3	3
MODIFIED RADICAL MASTOIDECTOMY	1	5
NASAL POLYPECTOMY	17	42
RADICAL FRONTAL SINUSOTOMY	3	3
RADICAL MASTOIDECTOMY	29	36
RADICAL MASTOIDECTOMY FOR CONGENITAL ATRESIA	2	1
REDUCTION OF NASAL FRACTURE	6	4
REVISION OF MASTOIDECTOMY	3	6
RHINOPLASTY	44	44
SIMPLE MASTOIDECTOMY	7	3
SPHENOIDOTOMY	1	4
STAPES MOBILIZATION	3	3
SMR OF NASAL SEPTUM	83	126
T & A	525	751
TURBINECTOMY	6	19



(A) MIDLINE INCISION STARTING IN CRICO-THYROID MEMBRANE

(B) EXPOSURE OF WEB

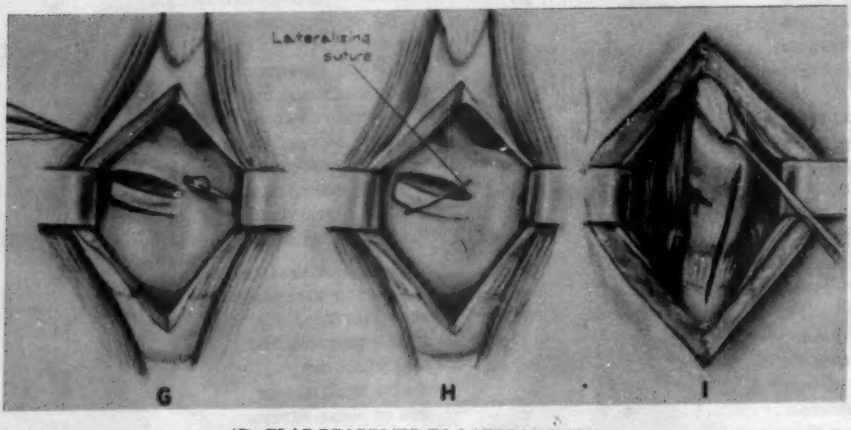
(C) WEB CUT



(D) FLAP OF MUCOSA CUT OVER RIGHT VOCAL CORD

(E) FLAP HINGE IN REGION OF VOCAL PROCESS

(F) MID TWO-THIRDS OF CORD RESECTED



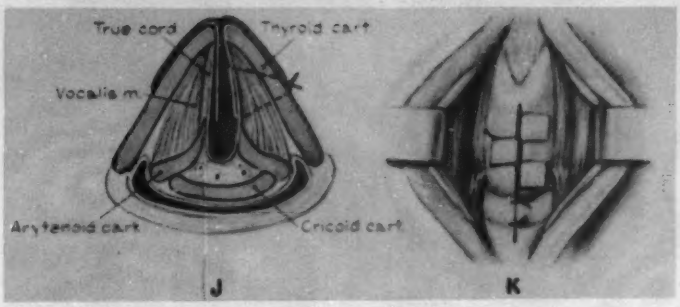
(G) FLAP REAPPLIED TO LATERAL WALL

(H) FLAP LATERALIZED WITH SUTURE PLACED IN REVERDIN NEEDLE

(I) SUTURE TIED OUTSIDE OF ALA CARTILAGE

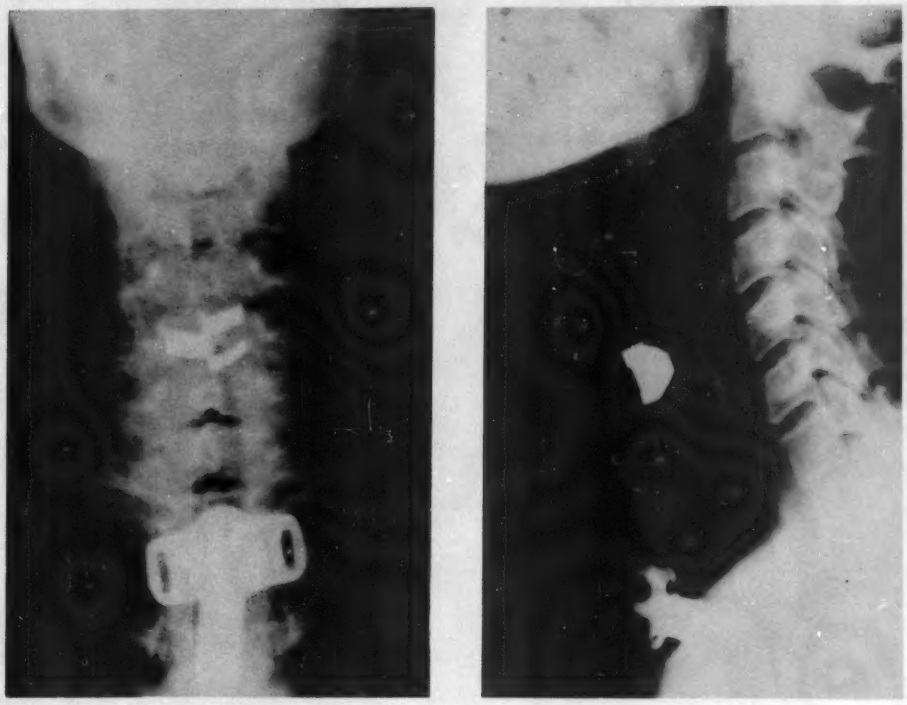
LARYNGEAL STENOSIS

USE OF MC NAUGHT'S KEEL



(J) PLACEMENT OF TANTALUM KEEL

(K) ANCHORING KEEL WITH LATERAL FLANGES DEEP TO RIBBON MUSCLES



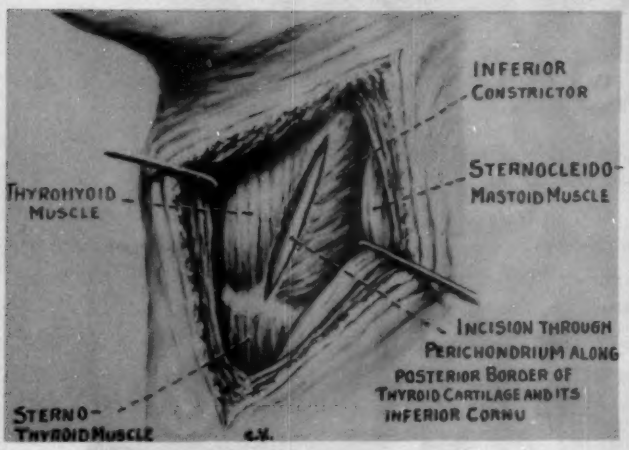
X-RAY SHOWING TANTALUM KEEL AND TRACHEOTOMY TUBE IN PLACE

The McNaught keel is used in cases where there is a web in the larynx of long standing and has many advantages and some disadvantages when compared with a core mold.

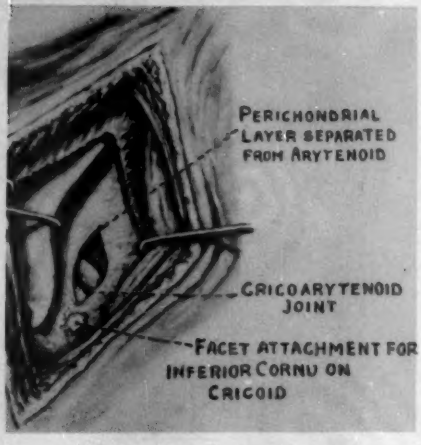
OPEN ARYTENOIDECTOMY

FOR

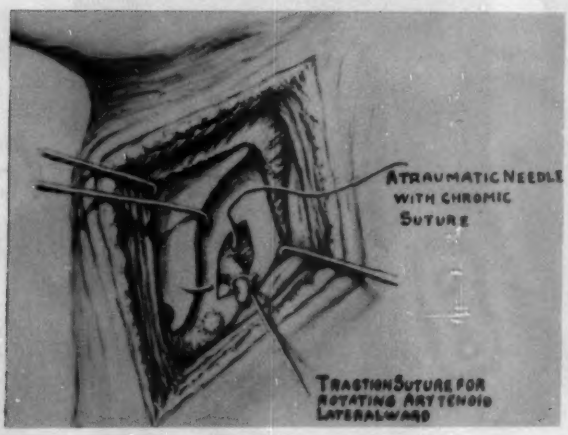
BILATERAL ABDUCTOR PARALYSIS



AN INCISION HAS BEEN MADE ALONG THE POSTERIOR BORDER OF THE LATERAL THYROID CARTILAGE AND THE INFERIOR CORNU DOWN TO AND THROUGH THE PERICHONDRIUM.



A TRACTION LIGATURE IS PLACED AROUND THE MIDPART OF THE ARYTENOID AND THE CARTILAGE ROTATED LATERALLY.



THE CORD IS DRAWN LATERALLY AND TIED AROUND THE INFERIOR CORNU OF THE THYROID CARTILAGE.

THE FACET LIKE JOINT BETWEEN THE INFERIOR CORNU AND THE CRICOID CARTILAGE IS THEN SEPARATED.

SURVEY OF 521 CASES BY NINETY SURGEONS	
OVERALL SUCCESSFUL CASES	88.3%
PRIMARY CASES SUCCESSFUL	91.0%
POST-OP. VOICE: ADEQUATE	75.0%
GOOD TO EXCELLENT	20.0%
POOR	5.0%

THE FACET LIKE JOINT BETWEEN THE INFERIOR CORNU AND THE CRICOID CARTILAGE IS THEN SEPARATED.

A TRACTION LIGATURE IS PLACED AROUND THE MUSCULAR PROCESS OF THE ARYTENOID AND THE CARTILAGE ROTATED LATERALLY.

THE CORD IS DRAWN LATERALLY AND TIED AROUND THE INFERIOR CORNU OF THE THYROID CARTILAGE

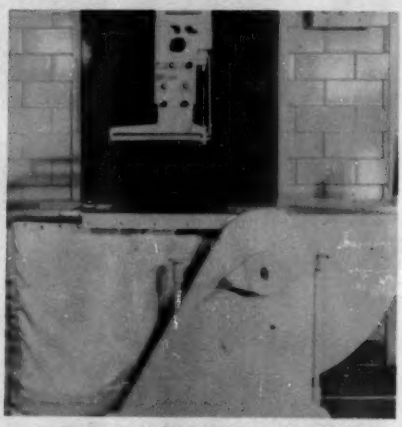
Open arytenoidectomy gives the surgeon a clear view of this field and is better adapted to the skill of many surgeons than more specialized intralaryngeal methods of relief of dyspnea from bilateral abductor paralysis.

ENDOSCOPY

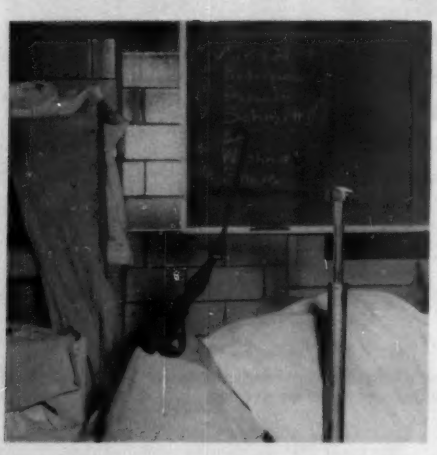
ANESTHESIA ROOM

CANOPIES WITH ALL INSTRUMENTS READY FOR INSTANT USE.

STUDENT GALLERY
GLOVE TABLE

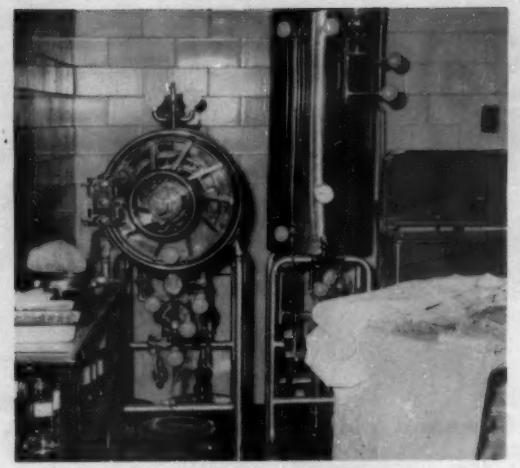


BIPLANE FLUOROSCOPE WITH PROTECTING FIBER GLASS APRON.



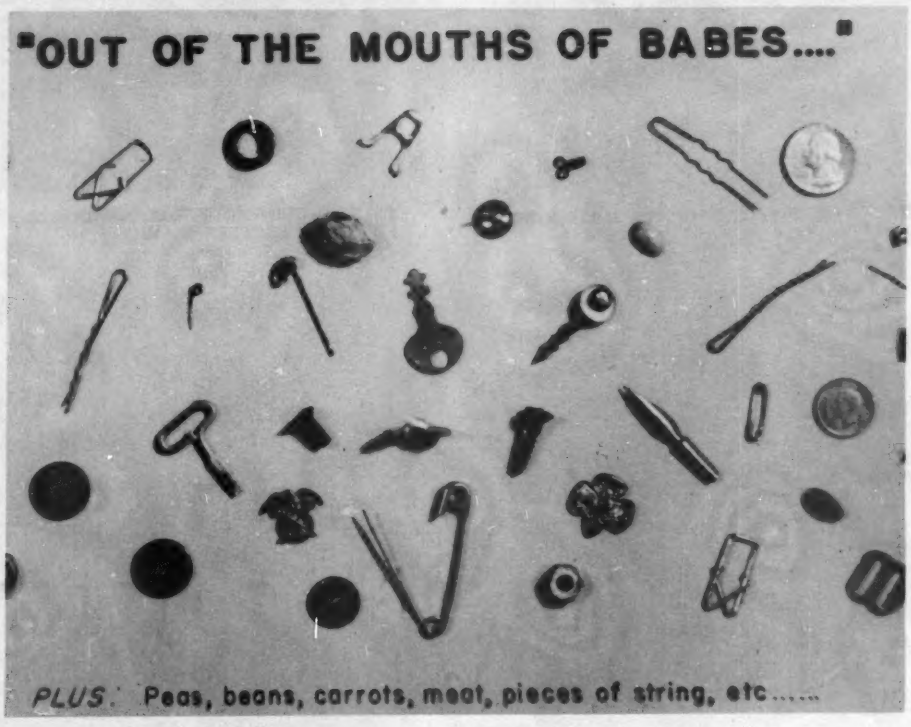
PATIENT IN POSITION FOR OPERATIVE WORK IN LARYNX USING SUSPENSION APPARATUS.

BIOPSY BEING TAKEN FROM LARYNX.

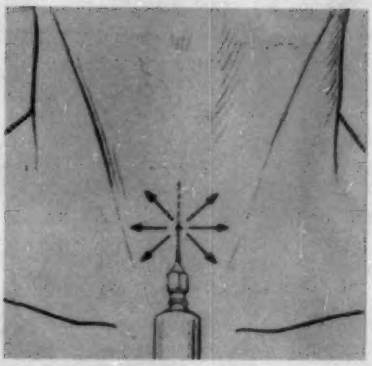


STERILIZERS IN WORK ROOM.

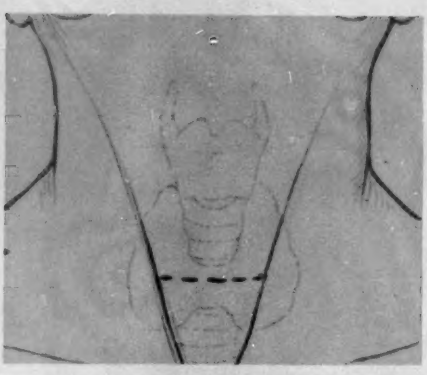
INSTRUMENT CLEANING.



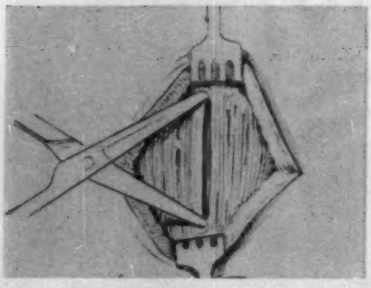
The layout in an endoscopy suite. Of importance is the green board and screen for use of the student gallery. Students can be far better instructed thusly than with a peek into a laryngoscope or bronchoscope. Photograph of representative foreign bodies recovered from air and food passages.



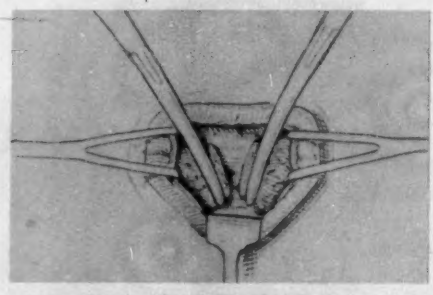
NOVOCAINE INFILTRATION.



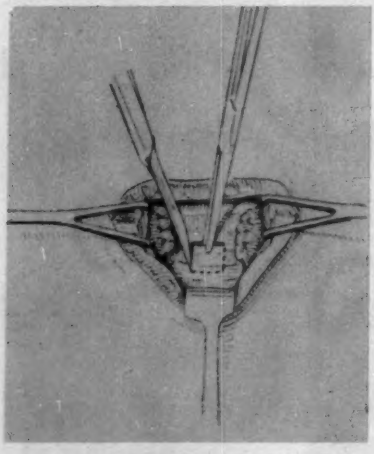
HORIZONTAL INCISION DOWN TO STRAP MUSCLES.



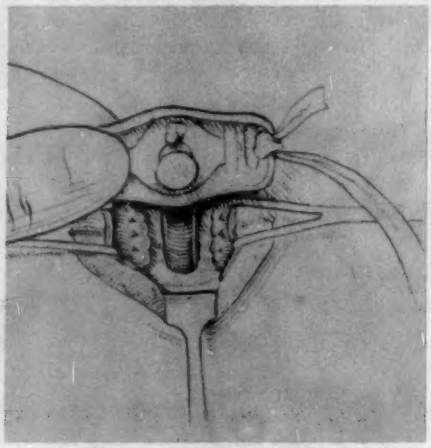
INCISION AND RETRACTION OF THE STRAP MUSCLES.



THYROID ISTHMUS IS PUSHED DOWNWARDS OR DIVIDED BETWEEN CLAMPS.



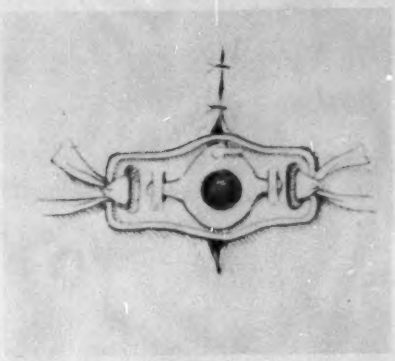
INCISION THROUGH 2nd AND 3rd CARTILAGENOUS RINGS AND REMOVAL OF PIECE OF CARTILAGE FROM 3rd RING FOR EASY RE-INSERTION OF TUBE.



INSERTION OF TRACHEOTOMY TUBE WITHOUT SKIN SUTURES.

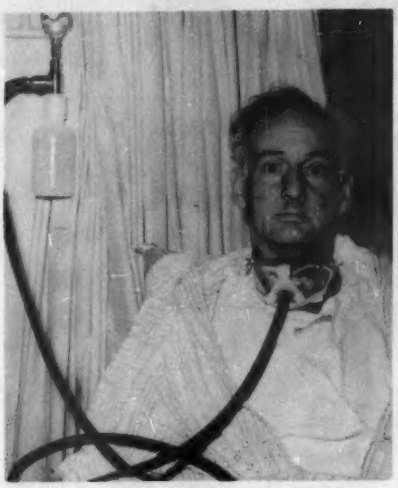
TRACHEOSTOMY

EMPHASIS ON ORDERLY OPERATION AND HORIZONTAL INCISION



VERTICAL INCISION FREQUENTLY RESULTS IN UNSIGHTLY SCAR. SUTURES MAY GIVE RISE TO MEDIASTINAL EMPHYSEMA.

TRACHEOTOMY SCAR FOURS WEEKS AFTER OPERATION.

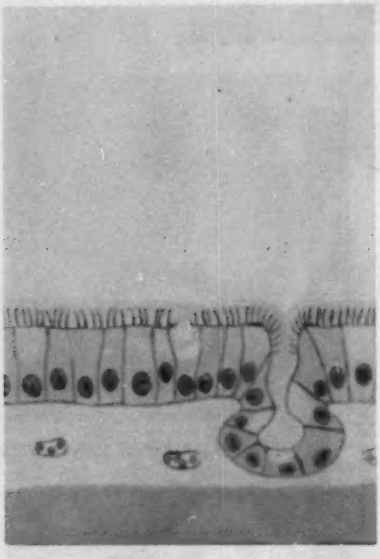


BEDSIDE VAPORIZER FOR TRACHEOTOMY. (BARACH)

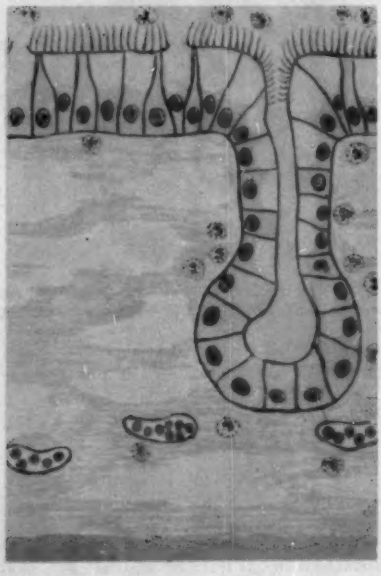
CROUPETTE FOR OXYGEN AND COLD VAPOR. IMPORTANT PROGRESS IN POST-TRACHEOTOMY CARE.

Horizontal incision tracheotomy, with emphasis on orderly tracheotomy, is featured because many physicians forget that tracheotomy should not be a last desperate procedure. With a bronchoscope in place, or with adequate time provided by a knowledge of the usual course of disease, or when tracheotomy is being done to provide tracheal toilet for a moribund postoperative patient, there is no need to mar the neck of either a male or female patient with an unsightly high vertical scar. At the bottom of the panel the application of cold vapor is depicted. This has been a most useful advance over hot steam for the care of patients with laryngotracheal disease.

Rx FOR RHINITIS



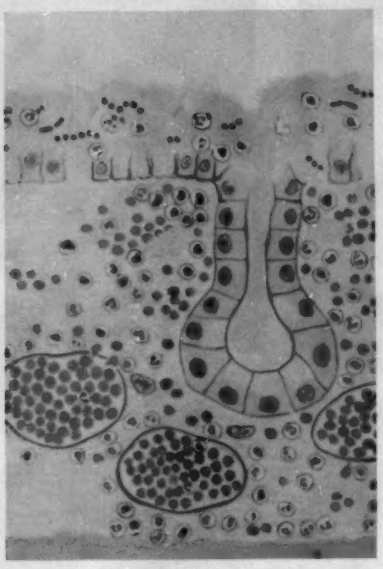
NORMAL NOSE



ALLERGIC NOSE

TRANSIENT RELIEF BY ANTI-HISTAMINES AND STEROIDS, SYSTEMICALLY AND LOCALLY.

ACUTE INFECTIOUS RHINITIS.



SECONDARY COMPLICATIONS CONTROLLED BY ANTIBIOTICS AND CHEMOTHERAPY.

LOCAL TREATMENT!!!

Antihistamines and steroids, such as cortisone, give temporary symptomatic relief from allergic troubles. This may prevent further complications and indicate that soon we may solve this problem for our patients. In the medical and surgical treatment of the infected nose there is still much to be learned. Antibiotics help the secondary infections. Local shrinkage, suction, and irrigation give temporary relief and patients now recover sooner than they used to, but the modern treatments for acute and chronic rhinitis and sinusitis still leave much to be desired. Overuse of nose drops often makes the patient worse.

TUMOR SURGERY

CHORDOMA OF NASOPHARYNX WITH EXTENSION INTO LEFT ORBIT.

OPERATION: EXCISION OF CHORDOMA IN LEFT MAXILLARY ETHMOIDAL AND SPHENOIDAL REGIONS WITH ENUCLEATION OF EYE AND SKIN GRAFTING.

SPECIMEN OF RADICAL NECK DISSECTION FOLLOWING SQUAMOUS CELL EPITHELIOMA OF NASOPHARYNX. FRESH SPECIMEN COVERED WITH PLATYSMA.

132 LYMPH NODES WERE FOUND IN THE SPECIMEN. THREE CONTAINED METASTASES: TWO IN THE UPPER END OF THE INTERNAL JUGULAR CHAIN, THE THIRD IN THE SPINAL ACCESSORY CHAIN. THE LYMPH NODE BEARING AREA WAS CLEARED.

CYLINDROMATOUS CARCINOMA OF RIGHT. SUB-MAXILLARY GLAND. PRE-OPERATIVE.

SPECIMEN FROM RIGHT HEMI-MANDIBULECTOMY. RADICAL RIGHT NECK DISSECTION AND LEFT UPPER NECK DISSECTION. THE OPERATION WAS DONE AS TREATMENT FOR RECURRENT CYLINDROMATOUS CARCINOMA OF THE SUB-MENTAL REGION.

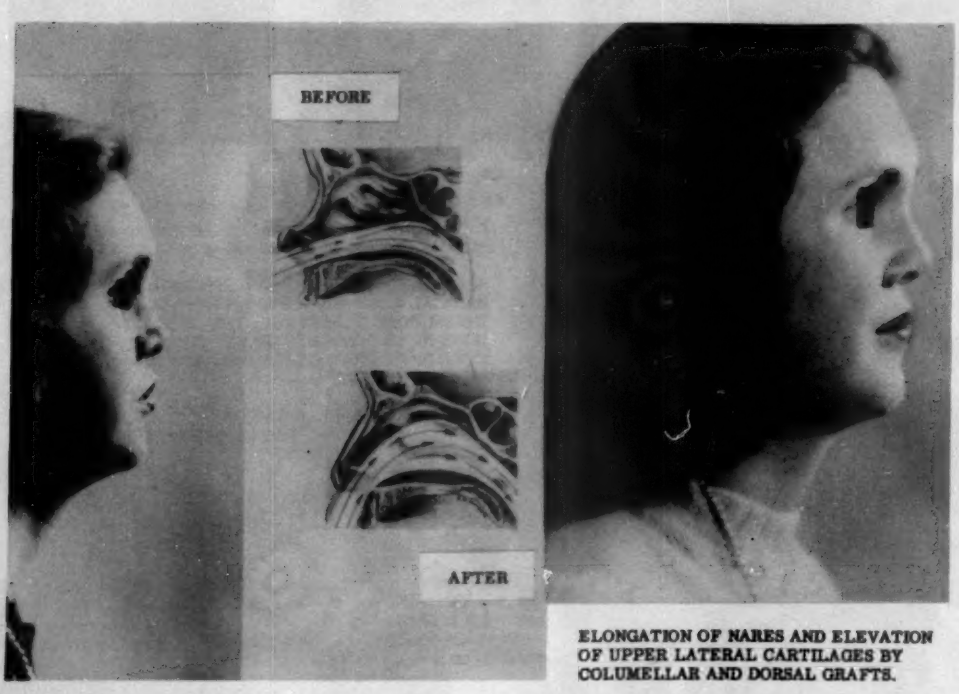
PATHOLOGICAL EXAMINATION OF THE SPECIMEN SHOWED THAT THE TUMOR WAS APPARENTLY COMPLETELY EXCISED.

Tumor surgery with en block dissection and removal of all lymph glands draining the area is featured here. The clearing technique facilitates search for involved lymph glands and helps in the after-treatment of such patients, especially when further surgery or x-ray treatments are indicated.

FUNCTIONAL RHINOPLASTY

LOWER SEPTAL DEVIATION AND EXTERNAL DEFORMITY CORRECTED BY RHINOPLASTY AND SUBMUCOUS RESECTION.

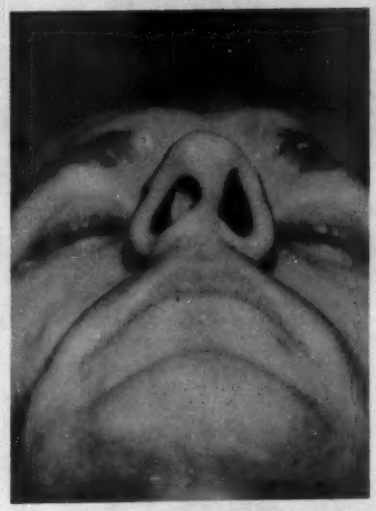
TWISTED NOSE



ELONGATION OF NARES AND ELEVATION OF UPPER LATERAL CARTILAGES BY COLUMELLAR AND DORSAL GRAFTS.

SADDLE NOSE

COMPLETE LUXATION OF SEPTUM AND DEVIATED NASAL TIP.



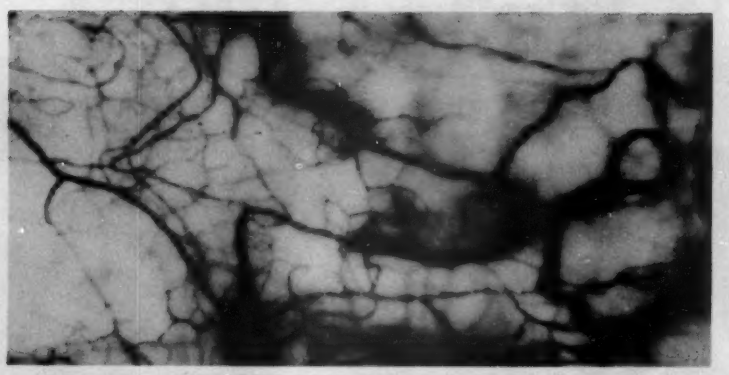
AFTER CORRECTION THROUGH SEPTAL PLASTIC AND RHINOPLASTY.

The provision of a better airway by plastic surgery in the nostrils is featured.

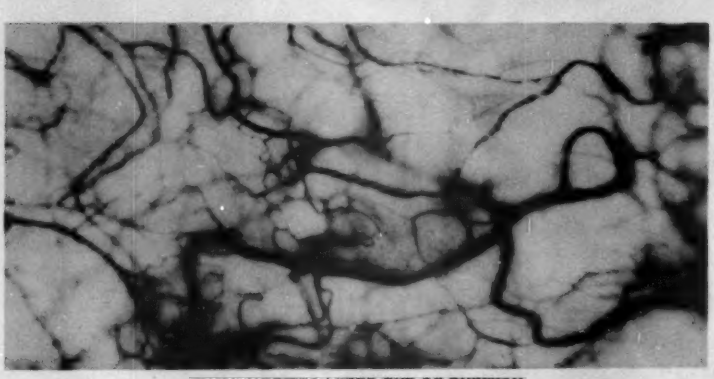
COLLAPSED ALA AND ANTERIOR DEVIATION OF SEPTUM.

AFTER REINFORCEMENT OF LOWER LATERAL CARTILAGE THROUGH GRAFT, AND SUBMUCOUS RESECTION, RE-ESTABLISHING AIRWAYS.

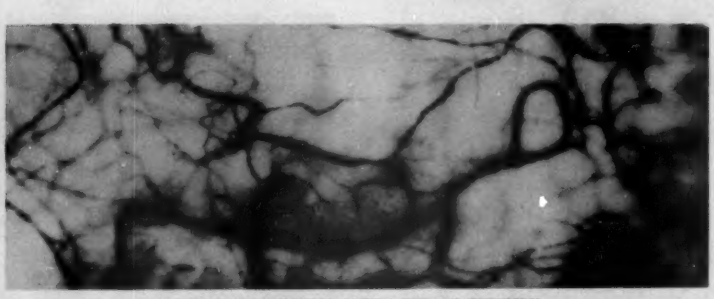
AFTER REINFORCEMENT OF LATERAL CARTILAGE THRO GRAFT, AND SUBMUCOUS RE RE-ESTABLISHING AIRWAYS



FIVE MINUTES AFTER BEGINNING OF INFUSION.



SEVEN MINUTES AFTER END OF INFUSION.



SEVENTEEN MINUTES AFTER END OF INFUSION.

NOTE:

(1) CHANGE IN DIAMETER OF VESSELS.
(2) DISAPPEARANCE OF RED BLOOD CELL LUMPING DURING INFUSION.
(3) RETURN OF LUMPING, BUT CLEARING OF SURROUNDING TISSUE AFTER INFUSION.

The medical treatment of Ménière's disease, Bell's palsy, and sudden deafness is indicated. Intravenous histamine and anticoagulation therapy are used with success in some cases.

CIRCULATION THERAPY

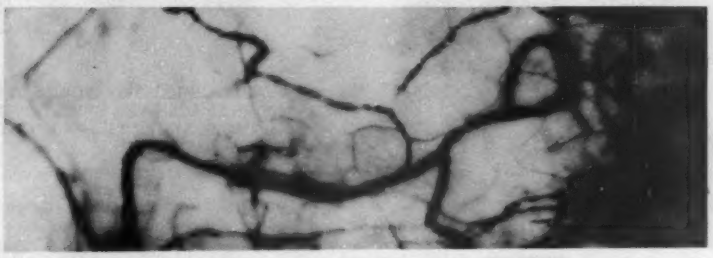
FOR

MENIERE'S DISEASE
BELL'S PALSY &
SUDDEN DEAFNESS

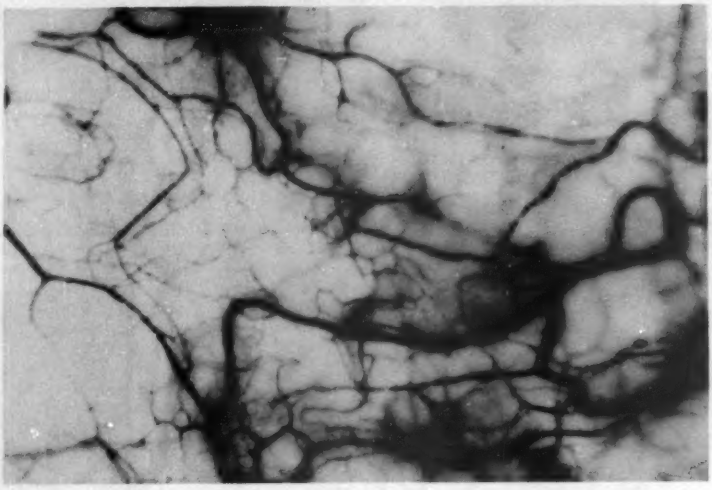
EACH PHOTOGRAPH REPRESENTS
2 mm. x 2.5 mm.

STANDARD TREATMENT FOR MENIERE'S DISEASE AND BELL'S PALSY IS NICOTINNIC ACID, 50-100 MG., 4 I.D., P.O., WITH TWO INFUSIONS A DAY OF 250 CC. OF 1/10TH OF 1% PROCAINE HYDROCHLORIDE, USING 5% DEXTROSE AS VEHICLE FOR TEN DAYS. THE INFUSION IS RUN THROUGH A #18 GAUGE NEEDLE AS RAPIDLY AS IT CAN BE TOLERATED. AVERAGE INFUSION OF 250 CC. TAKES ELEVEN MINUTES.

IF THE PATIENT DOES NOT HAVE A STRONG HEART INTRAVENOUS HISTAMINE IS TRIED AND IN BELL'S PALSY IT SEEMS TO SERVE AS WELL.

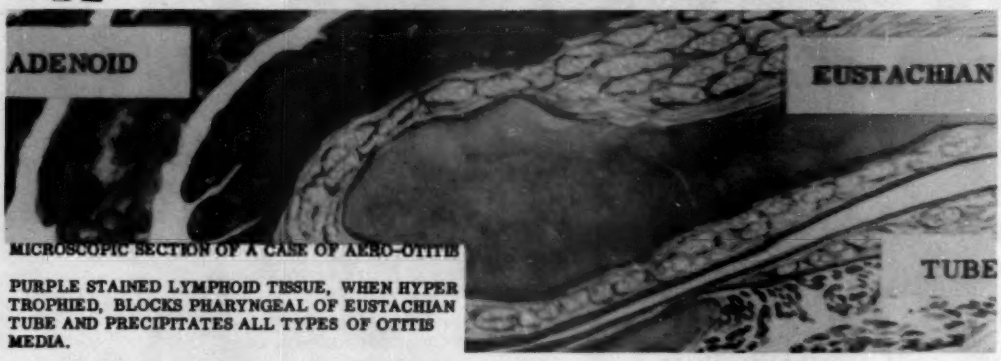


ONE MINUTE BEFORE BEGINNING OF INFUSION.



TWO MINUTES AFTER BEGINNING OF INFUSION.

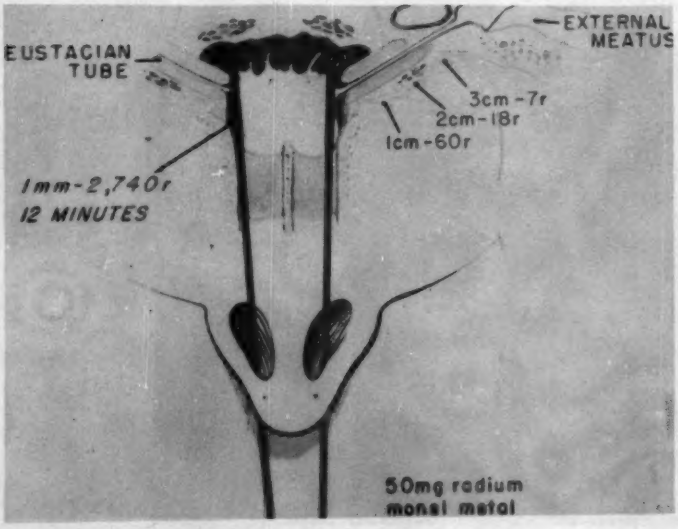
A RADIUM FOR RECURRENT OTITIS



MICROSCOPIC SECTION OF A CASE OF AERO-OTITIS

PURPLE STAINED LYMPHOID TISSUE, WHEN HYPER TROPHIED, BLOCKS PHARYNGEAL OF EUSTACHIAN TUBE AND PRECIPITATES ALL TYPES OF OTITIS MEDIA.

25 cm. RADIUM APPLICATOR (FOR PROTECTION OF OPERATOR) IN PLACE OVER HIGHLY SENSITIVE LYMPHOID TISSUE ABOUT THE PHARYNGEAL ORIFICE OF THE EUSTACHIAN TUBE.



HOW CROWE RADIUM APPLICATOR LOOKS FROM ABOVE. IN CROSS SECTION.

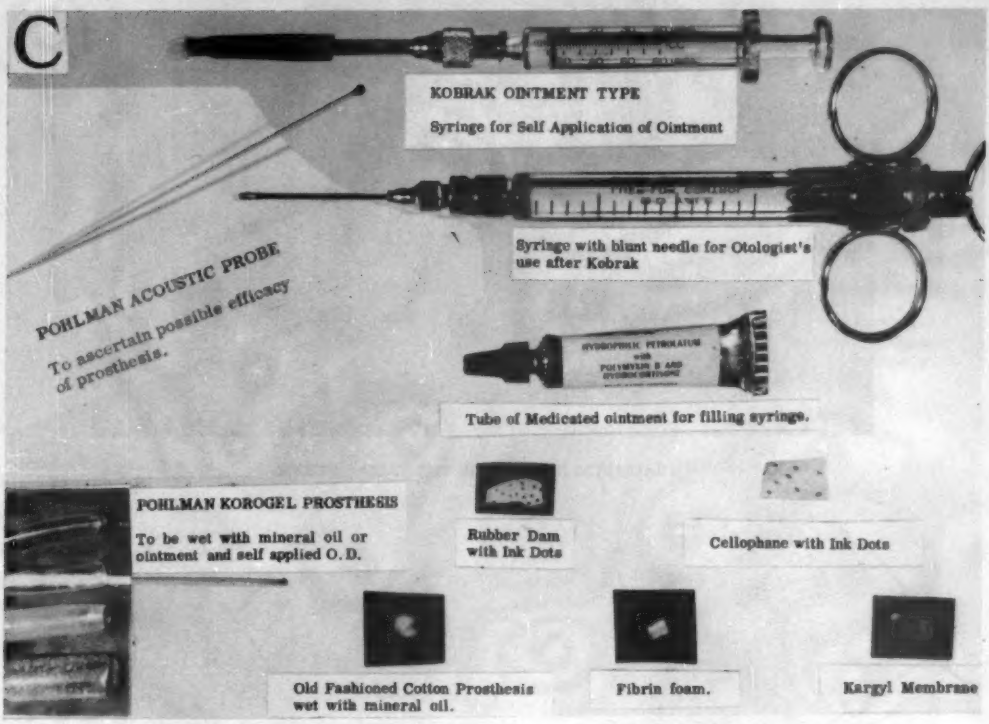
CALCULATED ROENTGEN DOSAGES AT VARIOUS DEPTHS FROM 0.3 mm. MONEL METAL APPLICATOR CONTAINING 50 mg. OF RADIUM SALT, WHICH DELIVERS ABOUT 25% GAMMA AND 75% BETA RADIATION.

B

POLYETHYLENE TUBES

Diameters: 0.5, 0.9 and 1.5 mm.

PROSTHESIS
FOR
CENTRAL PERFORATIONS OR RADICAL MASTOIDECTOMIES


C
KOBRAK OINTMENT TYPE
Syringe for Self Application of Ointment
Syringe with blunt needle for Otologist's use after Kobrak
POHLMAN ACOUSTIC PROBE
To ascertain possible efficacy of prosthesis.
Tube of Medicated ointment for filling syringe.
POHLMAN KOROGEL PROSTHESIS
To be wet with mineral oil or ointment and self applied O. D.
Rubber Dam with Ink Dots
Cellophane with Ink Dots
Old Fashioned Cotton Prosthesis wet with mineral oil.
Fibrin foam.
Kargyl Membrane

A, excess lymphoid tissue about the nasopharyngeal mouth of the Eustachian tube has long been known to be a factor in otitis media, especially recurrent otitis media and aero-otitis. The Crowe method is diagrammatically shown with indication of the dosage at intervals from the radium applicator.

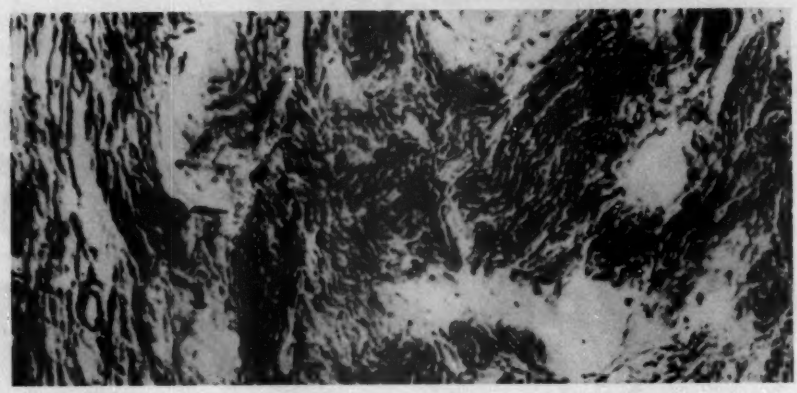
B, for chronic secretory otitis media, paracentesis with aspiration and the insertion of a 1.5 cm. long piece of polyethylene tubing help to equalize the pressure of the middle-ear end of the Eustachian tube and often eliminate the necessity for multiple myringotomies (Armstrong, B. W.: A. M. A. Arch. Otolaryng. **59**:653 [June] 1954).

C, prostheses for the deafness of chronic otitis and for dry perforations are often most rewarding. New and old-fashioned ones are shown. From the patient's standpoint self-application of a medicated hydrophilic petrolatum is easy and helpful. It improves the hearing when correctly installed according to the method of Kobrak in the niches of the round and the oval window and keeps down the infection at the same time. If ointment alone does not improve the hearing sufficiently the correct size of Pohlman prosthesis on top of it often will.

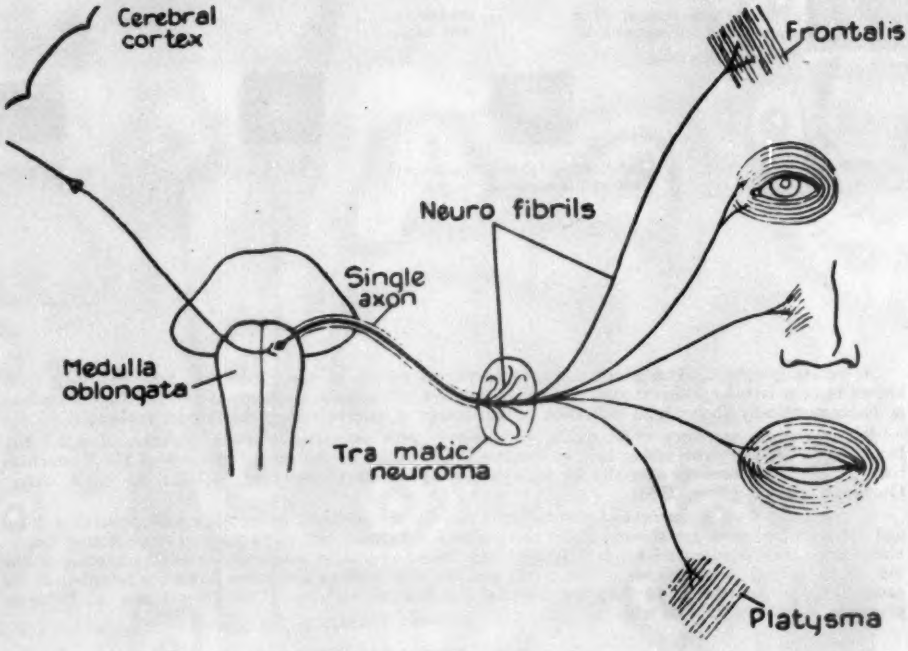
FACIAL NERVE DECOMPRESSION

FOR

BELL'S PALSY
TRAUMATIC INJURY
GRAFTING



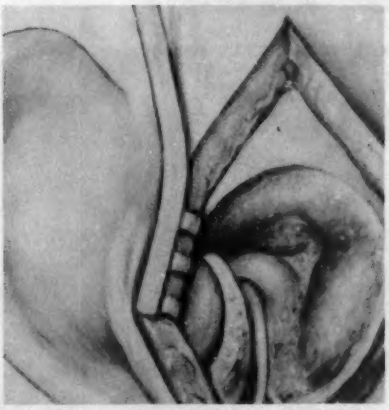
TRAUMATIC NEUROMA IN THE FACIAL NERVE.



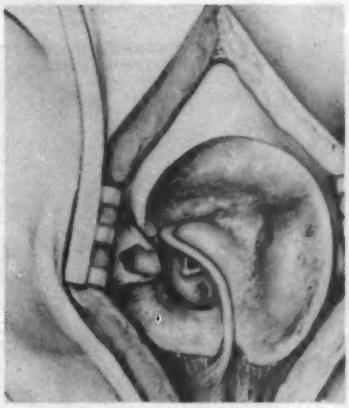
MECHANISM OF ASSOCIATED MOVEMENTS WHICH INVARIABLY FOLLOW TRAUMA TO THE FACIAL NERVE.

PALSY FOLLOWING REMOVAL OF A CHOLESTEATOMA SURROUNDING THE FACIAL NERVE, RIGHT.

SAME PATIENT TWO YEARS AFTER DECOMPRESSION. NOTE INCREASED MOVEMENT OF RIGHT SIDE OF FACE.



DECOMPRESSION OF NERVE VII THROUGH SIMPLE MASTOID APPROACH. USED FOR RECURRENT, PERSISTENT BELL'S PALSY AND FRACTURE OF THE SKULL, WHERE THE INNER EAR IS NOT INVOLVED.



DECOMPRESSION OF FACIAL NERVE FROM GENICULATE GANGLION TO STYLOID MASTOID FORAMEN.

Traumatic or infectious injury severe enough to cause Wallerian degeneration results in a neuroma. This results in associated movements after repair. The mechanism and end-result of increased motion on the recovered side are shown. Also, two dissections: one showing decompression of the nerve by the simple mastoidectomy technique as used in Ball's palsy; the other complete decompression, such as usually is necessary in traumatic lesions of the nerve.

TYMPANOPLASTY

FOR

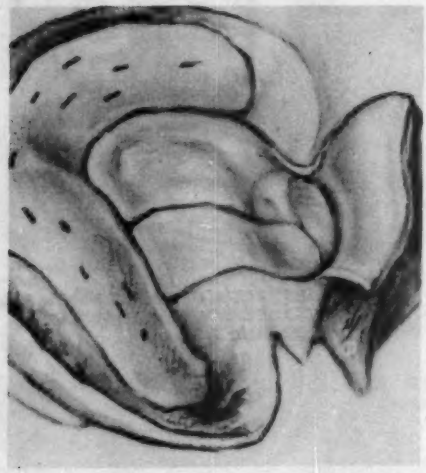
CHRONIC OTITIS MEDIA SEQUELAE
OR AFTER RADICAL MASTOIDECTOMY

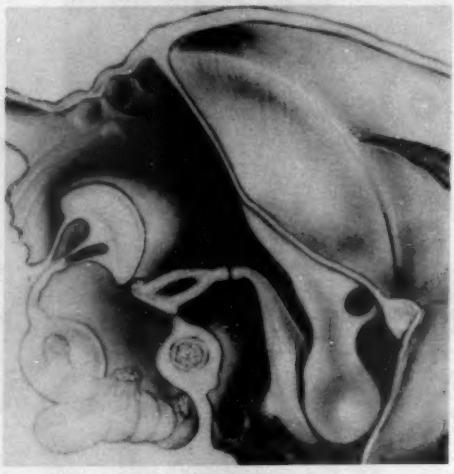
PLASTIC RECONSTRUCTION OF THE SOUND CONDUCTING APPARATUS OF THE MIDDLE EAR.

PREREQUISITES:

COMPLETE ERADICATION OF INFECTION.
EUSTACHIAN TUBE PATENCY.
NORMAL STAPES AND ROUND WINDOW MEMBRANE MOTILITY.
GOOD INNER EAR FUNCTION.

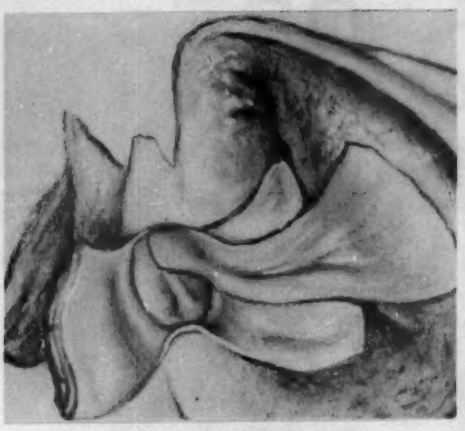
SEMI-DIAGRAMMATIC VERTICAL SECTION THROUGH THE EAR, SHOWING DRUM MEMBRANE, OSSICLES, WINDOWS, EUSTACHIAN TUBE, ETC.





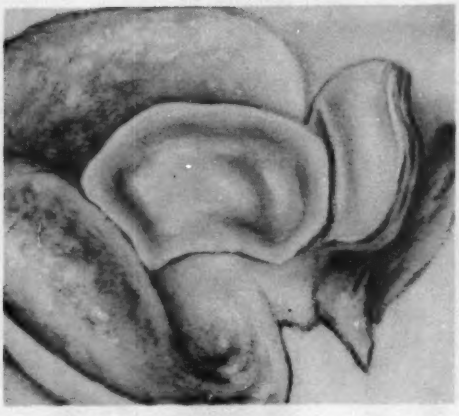
RECONSTRUCTION OF THE ENTIRE TYMPANIC CAVITY IN A CASE WITH POSTEROSUPERIOR MARGINAL PERFORATION OF THE DRUM; CHAIN OF OSSICLES PRESERVED AND FUNCTIONING.

MODIFIED RADICAL MASTOIDECTOMY WITH PRESERVATION OF A THIN PORTION OF THE BRIDGE. FLAP TURNED UPWARD, FREE FULL THICKNESS GRAFT CLOSING THE PERFORATION AND COVERING THE LOWER PART OF THE FACIAL RIDGE.



RECONSTRUCTION OF THE ENTIRE TYMPANIC CAVITY IN A CASE WITH POSTEROSUPERIOR MARGINAL PERFORATION OF THE DRUM; CHAIN OF OSSICLES PRESERVED AND FUNCTIONING.

MODIFIED RADICAL MASTOIDECTOMY WITH REMOVAL OF THE BRIDGE, THOROUGH REMOVAL OF THE GRANULATIONS, FORMATION OF A TYMPANOMEATAL FLAP, LENGTHWISE SPLITTING OF THE FLAP INTO THE PERFORATION WITH FORMATION OF AN UPPER AND LOWER PORTION OF THE FLAP, FREE FULL THICKNESS SKIN GRAFT INTERPOSED BETWEEN THE TWO PORTIONS, CLOSING THE PERFORATION AND COVERING THE INCUS AND MALLEUS.



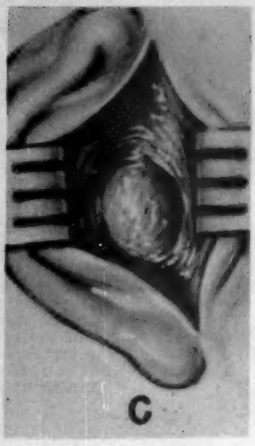
COMPLETE LOSS OF DRUM MEMBRANE AND OF INCUS AND MALLEUS, STAPES PRESERVED.

RADICAL MASTOIDECTOMY FOLLOWED BY RECONSTRUCTION OF THE LOWER PART OF THE TYMPANIC CAVITY BY MEANS OF A FREE FULL THICKNESS SKIN GRAFT FROM THE RETROAURICULAR REGION, WHICH IS IN CONTACT WITH THE HEAD OF THE STAPES (FOR COLUMELLAR EFFECT).

Wullstein, Zollner, Baron, and others are providing new hope that patients with adhesive deafness, with or without radical cavities, may be helped. Various techniques are shown, but this surgery must be carefully tailored to the individual case.

RELIEF OF MEATAL ATRESIA

TWO STAGE PROCEDURE FOR RELIEF OF DEAFNESS FROM CONGENITAL ANAMOLIES OF THE EAR



(A) PRE-OPERATIVE APPEARANCE OF RIGHT AURICLE.

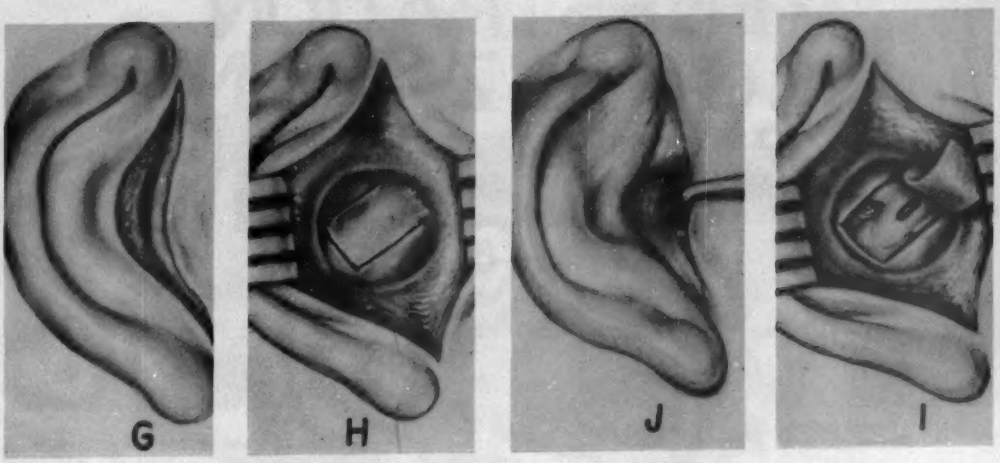
(B) ENDAURAL INCISION.

(C) INCISION DOWN TO BONE SHOWING NO EVIDENCE OF AN EXTERNAL CANAL.

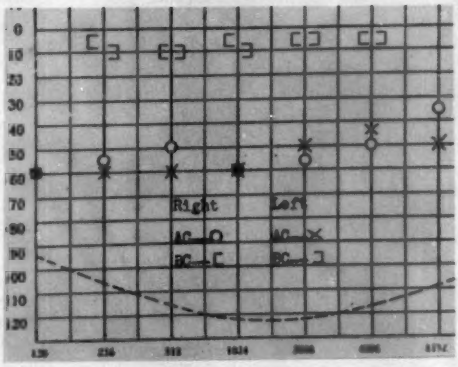
(D) MASTOID CELLS EXENTERATED, EXPOSING MALFORMED INCUS AND MALLEUS.

(E) HORIZONTAL CANAL SKELETONIZED.

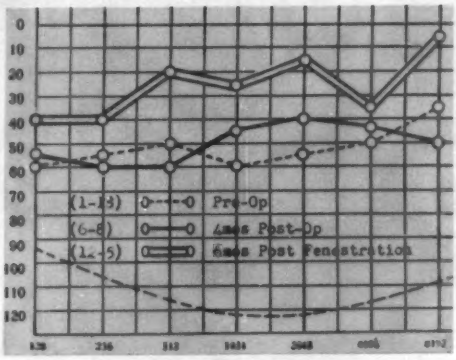
(F) PLACEMENT OF SKIN GRAFTS.



(G) ENDAURAL APPROACH.
(H) EXPOSURE OF CAVITY AND CUTTING OF FLAP.
(I) FENESTRATION OF HORIZONTAL CANAL.
(J) POST-OPERATIVE APPEARANCE OF EXTERNAL CANAL.



PRE-OPERATIVE AUDIOGRAM.



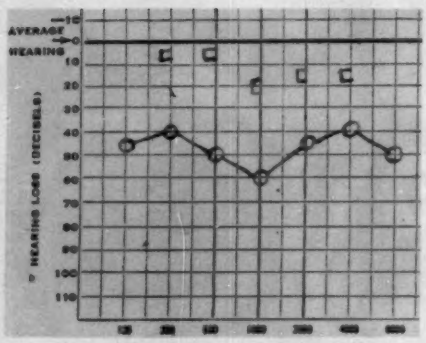
COMPARATIVE AUDIOGRAM OF RIGHT EAR.
BEFORE OPERATION AND AFTER EACH OPERATIVE PROCEDURE.

FENESTRATION MOBILIZATION

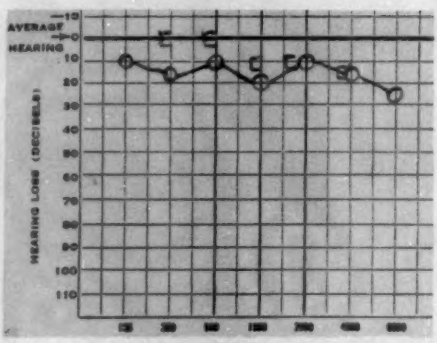
FOR

DEAFNESS CAUSED BY ANKYLOSIS OF THE STAPES

EXPOSURE OF INCUS, POSTERIOR CRUS OF STAPES AND STAPEDIUS TENDON BY LIFTING THE DRUM AND ADJACENT MEATAL SKIN AFTER THE METHOD OF ROSEN, FOR MOBILIZATION OF STAPES.

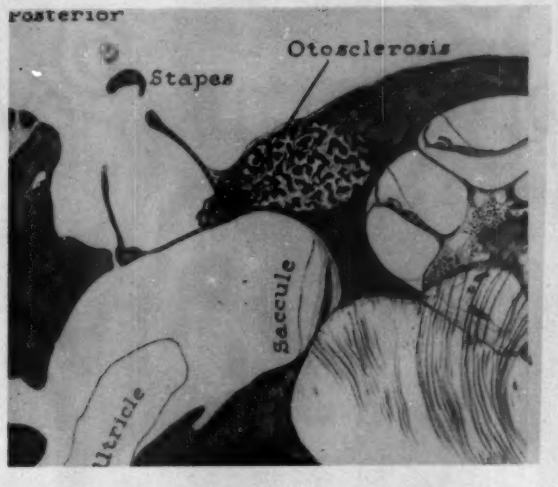


PRE-OPERATIVE AUDIOGRAM.



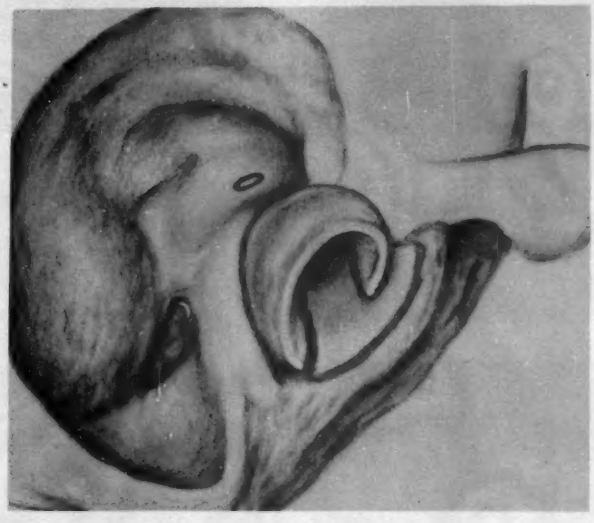
POST-MOBILIZATION AUDIOGRAM.

The most stimulating part of modern otolaryngology, to the otologic surgeon, is the development of the fenestration operation and mobilization procedures for ankylosis of the stapes. These are best performed under magnification of $\times 4$ to $\times 10$. The latter operation, although simple for the patient, is much the more difficult of the two. Otologic surgery has been much advanced by the introduction of proper lighting, magnification, and dental instruments, particularly the dental engine.

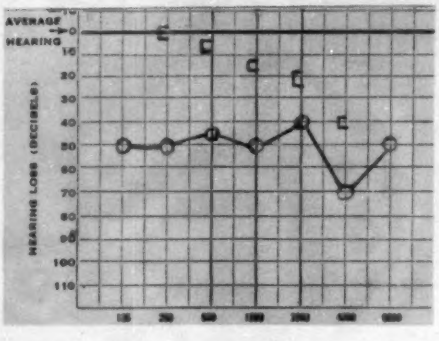


DRAWING OF MICROSCOPIC SECTION SHOWING BONY OTOSCLEROTIC GROWTH IN THE ANTERIOR BORDER OF THE OVAL WINDOW.

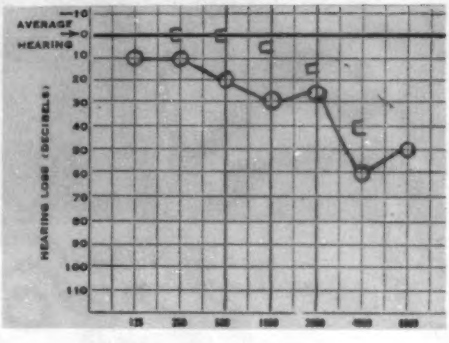
STERILE PLASTIC SHIELD OVER MICROSCOPE IN USE IN OPERATING THEATER.



FENESTRATION OF THE AMPULLATED PORTION OF THE HORIZONTAL SEMI-CIRCULAR CANAL.



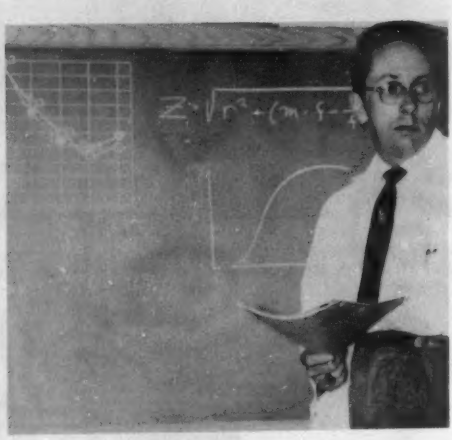
PRE-OPERATIVE AUDIOGRAM.



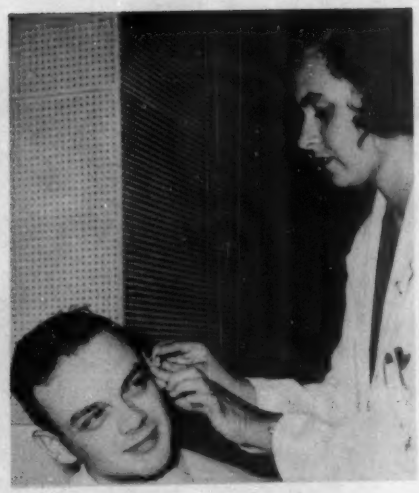
POST-FENESTRATION AUDIOGRAM.

AUDIOLOGY & SPEECHPATHOLOGY

RESEARCH.



TEACHING.



EAR IMPRESSIONS.

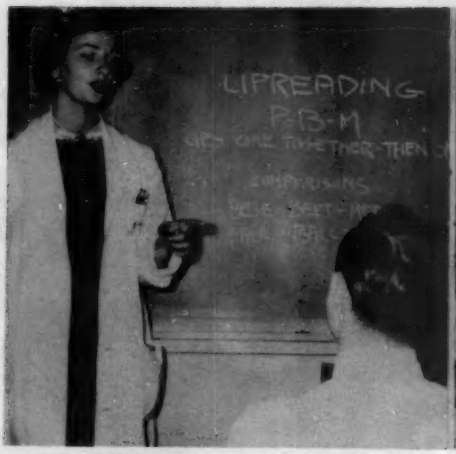
HEARING AID SELECTION.

PURE TONE AUDIOMETRY.

SPEECH AUDIOMETRY.

SPEECH & LANGUAGE EVALUATION.

AUDITORY TRAINING.



SPEECH THERAPY.

LIP READING.

ESOPHAGEAL VOICE TRAINING.

Pages 30, 31, and 32 show the activities in the Hearing and Speech Section of the Department of Otolaryngology. These include testing, teaching, and rehabilitation for all age groups. Reduction in the size of hearing aids, 1930, 1940, and 1955, is dramatically shown.

CARBON HEARING AID
1930

VACUUM TUBE HEARING
AID 1940

TRANSISTOR HEARING
AID 1955

Deafness and Vertigo from Head Injury

HAROLD F. SCHUKNECHT, M.D., Detroit
and
RODERICK C. DAVISON, M.D., Winnipeg, Man., Canada

Deafness and vertigo are common complaints of the patient with head injury. By studying the clinical findings in human patients and correlating them with the findings of animal experiments, we have concluded that these symptoms are due, for the most part, to injury of the membranous labyrinth.

I. CLINICAL MANIFESTATIONS

The auditory and vestibular symptoms following head injury can be classified according to the following types of labyrinthine damage: A. longitudinal fracture of the temporal bone; B. transverse fracture of the temporal bone; C. labyrinthine concussion.

A. Longitudinal Fracture of the Temporal Bone

Typically, a longitudinal fracture of the temporal bone is a linear fracture through the floor of the middle cranial fossa, which lies parallel and adjacent to the anterior margin of the petrous pyramid. It extends medially from the region of the Gasserian ganglion to the middle ear and mastoid air cells laterally. On roentgenograms the fracture usually can be seen as a linear streak in the squamous portion of the temporal bone and the mastoid air cells are clouded by the presence of blood or cerebrospinal fluid. About 80% of temporal-bone fractures are of the longitudinal type.[11] The experiments of Proctor and associates [34] and Gurdjian and Lissner [16] have shown that longitudinal fractures more commonly occur from blows to the parietal and temporal regions of the skull than from blows to the occipital or frontal regions. Characteristically, the fracture line traverses the annulus tympanicus, creating a laceration of the tympanic membrane and bleeding from the ear. A step-like deformity of the external canal can occur when the fracture extends through it. Facial weakness or paralysis occurs in less than 25% of cases and usually is temporary.[44] Cerebrospinal otorrhea occurs in some cases.

Histological study of human temporal bones following longitudinal fracture has revealed in some instances subluxation of the incudostapedial joint. In severe injury rupture of the annular ligament of the stapes footplate may also occur.[19]

Damage to the eardrum and ossicles, as well as the presence of blood in the external auditory canal and middle ear, creates a conductive type of hearing loss. Audiometric study reveals a depression of air-conduction thresholds throughout the auditory spectrum. Tuning-fork tests show better hearing by bone conduction than by air conduction (negative Rinne test). When the handle of the tuning fork is placed firmly on the midline of the head the tones are best heard in the fractured ear (Weber test). For routine testing we utilize a 512 cps tuning fork; however, additional forks often are used to gain information over a wider frequency range. Often there is superimposed upon the conductive deafness a high-tone perceptive type

Accepted for publication Feb. 14, 1956.

From the Department of Otolaryngology of the Henry Ford Hospital.

deafness.* It is manifested, of course, by a depression of bone-conduction thresholds and may vary from a small dip for the 4000 cps frequency to a severe loss involving a wide range of frequencies.[26] This perceptive type deafness is discussed in detail under the heading "Labyrinthine Concussion."

As the blood is absorbed and the injury of the eardrums and ossicles heals the hearing improves.[14] The perceptive portion of the deafness usually is partially reversible, and so bone-conduction thresholds can be expected to improve during the first three or four weeks following injury.† In many cases, however, part of the perceptive loss persists, and it is particularly characteristic for a threshold loss to persist for frequencies of 4000 cps and above.

Patients having had a longitudinal fracture frequently experience transient vertiginous attacks for a period of days or months. An attack may be described as a feeling of unsteadiness or as a true spinning sensation, but in either case the episodes usually are precipitated by postural changes of the head. An attack usually lasts but a few seconds and is unaccompanied by nausea and/or vomiting. In severe head injuries this symptom may be due, in part, to involvement of the vestibular neural pathways. We believe, however, that it is more commonly the result of damage to the vestibular labyrinth. Evidence in support of this concept is presented later in this paper.

The typical clinical features of longitudinal fracture of the temporal bone are revealed in the following two case histories:

Case 1.—A 23-year-old electrician received a compression injury of the head in a press used to form automobile fenders. He was unconscious for about 10 minutes and bled from both ears. He then complained of hearing loss in both ears and brief dizzy spells on postural change, such as when rising to the sitting position or when rolling over in bed. Roentgenograms revealed multiple skull fractures, including a longitudinal fracture of both temporal bones. Otological examination two days after injury revealed dried blood in both external auditory canals. There were healing lacerations and bluish discoloration of both tympanic membranes, indicating blood-filled middle ears. The Rinne test was bilaterally negative (BC greater than AC) with the 512 cps tuning fork. Two weeks later the

* Perceptive deafness refers to all hearing losses not caused by pathology of the sound-conducting system but makes no distinction between the lesions of the sense organ, cochlear nerve, or higher auditory pathways.

† References 2, 14, and 44.

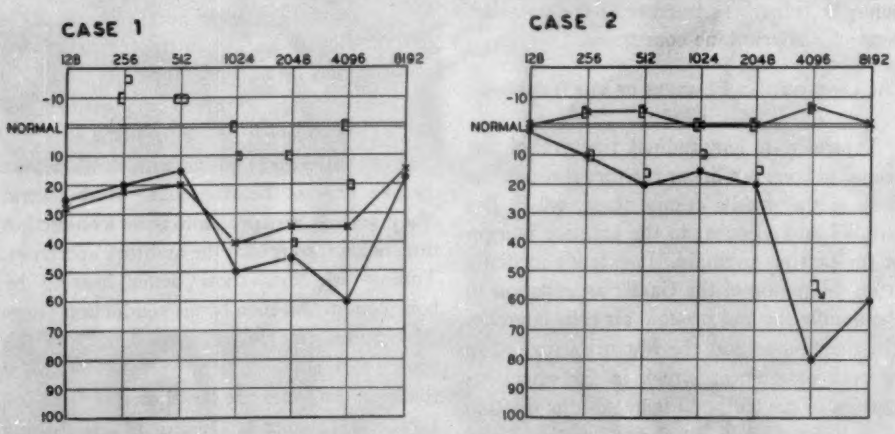


Fig. 1.—Longitudinal fractures of the temporal bone (bone conduction for right and left ears; air conduction for right and left ears): *Case 1*, audiogram two weeks after bilateral longitudinal temporal-bone fractures. There is a bilateral conductive deafness due to blood-filled middle ears and, in addition, a perceptive deafness in the right ear which reaches a maximum loss of 40 db. for 2048 cps. *Case 2*, audiogram four months after longitudinal fracture of the right temporal bone. The depressed bone-conduction thresholds reveal a mild perceptive deafness in the right ear for frequencies from 256 to 2048 cps and a severe deafness for 4096 and 8192 cps.

tympanic membranes were still discolored and the audiogram showed a combined conductive-perceptive deafness in both ears (Fig. 1, Case 1). Bluish-yellow fluid was aspirated from both middle ears at this time and again one week later. Four weeks following injury the tympanic membranes appeared normal and the conductive element in the hearing loss had disappeared, although the perceptive element remained. The episodes of postural vertigo subsided gradually over a three-month period.

CASE 2.—A 29-year-old male salesman was involved in an automobile accident, following which he was unconscious for an hour and had a discharge of blood and cerebrospinal fluid from the right ear. Roentgenograms revealed a longitudinal fracture of the right temporal bone. On regaining consciousness he complained of hearing loss in the right ear. The next day otological examination revealed a serohemorrhagic fluid in the right ear canal and a moderately severe combined conductive-perceptive hearing loss in the right ear. He complained for four months of transient episodes of unsteadiness following sudden postural changes. An audiogram taken four months after injury is shown in Figure 1, Case 2, and shows a persistent perceptive hearing loss, worse for high frequencies.

B. TRANSVERSE FRACTURE OF THE TEMPORAL BONE

A transverse fracture of the temporal bone occurs perpendicular to the long axis of the petrous pyramid and is most likely to result from blows to the occiput.‡ In about 60% of the cases the fracture can be demonstrated roentgenographically in either the submento-vertex or Stenver's position of the skull.[14] In about 50% of the cases the facial nerve is lacerated, and the resultant facial palsy sometimes is permanent. Bleeding from the ear is rare although hematotympanum (blood in the middle ear) is a frequent finding. The tympanic membrane is at first dark blue, later becoming light blue, and then rose red, before complete resolution occurs, in two to four weeks.[85] Cerebrospinal fluid may continue to fill the middle ear after the blood is absorbed. In such cases the cerebrospinal fluid may drain through the Eustachian tube into the nasopharynx.[9] The transverse fracture traverses the vestibule of the inner ear, causing extensive destruction of the membranous labyrinth and usually complete loss of cochlear and vestibular function.[48] Severe vertigo exists for several days and is accompanied by nausea and vomiting. It subsides gradually over a period of two to three weeks, although unsteadiness and a tendency to sway to the side of the involved ear when walking may persist for months. A mild spontaneous nystagmus with the quick component to the opposite side may exist for many months. Some cases of incomplete loss of auditory and vestibular function following transverse fracture of the temporal bone have been reported.§ In such cases there may have been an isolated or incomplete fracture of the labyrinth capsule. Isolated fractures of the cochlea and internal meatus have been described in autopsy specimens.‖

Case histories of two patients with transverse fracture follow:

CASE 3.—A 29-year-old male physician was injured in an automobile accident, following which he was unconscious for five days. At no time was there bleeding from his ears or facial palsy. After he regained consciousness he suffered from severe vertigo and nausea for several days and noticed profound deafness in his right ear. Skull roentgenograms demonstrated a transverse fracture of the right petrous pyramid. Vertigo subsided gradually during the subsequent three or four weeks, but for several months he noticed a tendency to sway to the right when walking. When he was last examined, two years after the accident, there was no caloric response to 5 cc. of ice water in the right ear, whereas in the left ear there was a response of 1 minute 30 seconds (normal) to 5 cc. of water at 80 F.¶

He could not understand words shouted into the right ear when the left ear was masked with the Bárány noise box. Audiometric study revealed a profound deafness in the right ear. In the left ear there was a slight perceptive deafness for very low frequencies and a severer loss for high frequencies, with the peak loss at 4096 cps (Fig. 2, Case 3).

‡ References 16 and 34.

§ References 12, 13, 18, and 25.

‖ References 16 and 36.

¶ All tests were performed with the patient in the sitting position with the head tilted back at a 60 degree angle. The water was injected against the eardrum from a glass syringe through a 20-gauge needle at a rate of 1 cc. per second. Time was recorded from the end of injection of water to the end of nystagmus, by use of Frenzel glasses.

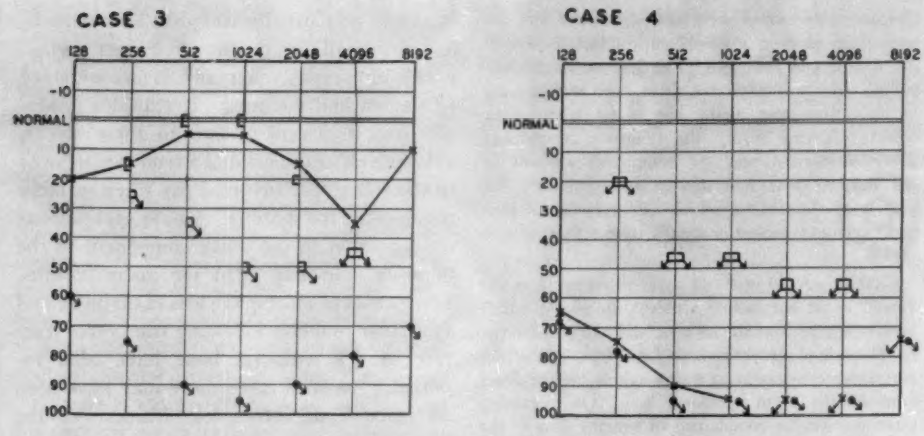


Fig. 2.—Transverse fractures of the temporal bone. The arrows indicate that the tones were not heard at the maximum intensities available with the audiometer: *Case 3*, there is total deafness for air- and bone-conducted tones on the right and a 4096 cps dip of the perceptive type on the left. *Case 4*, audiogram seven weeks after occipital head injury. Total deafness on the right. Tones from 128 to 1024 cps faintly perceptible at high intensities on the left.

Case 4.—A 34-year-old male interior decorator was struck with a fist during an altercation. He fell, striking the occiput of his head on the sidewalk, but did not lose consciousness. There was no bleeding from his ears and no facial palsy. He went home immediately and complained to his wife of unsteadiness, deafness, and roaring in his ears. He was first seen by us seven weeks later, at which time audiometric studies revealed total deafness in the right ear and almost total deafness in the left (Fig. 2, Case 4). There was no spontaneous nystagmus. Caloric tests with 5 cc. of ice water gave no response in the right ear and only faint nystagmoid movements lasting about 10 seconds in the left ear. He had very little dizziness but felt constantly unsteady. He was slightly ataxic and walked with a wider base than normal. Roentgenograms revealed a fracture of the occipital bone and a transverse fracture of the right temporal bone. Although it was not demonstrable by roentgenogram, we believe that there probably also existed a transverse fracture of the left temporal bone, to explain the severe loss of both cochlear and vestibular function in the left ear.

C. Labyrinthine Concussion

Labyrinthine concussion may be described as perceptive deafness and vertigo resulting from a blow to the head without fracture of the bony labyrinthine capsule. This injury is commonest in the ear of a longitudinally fractured temporal bone; secondly, in the ear opposite a temporal-bone fracture, and, thirdly, in head injury with no evidence of skull fracture. To produce labyrinthine concussion a head injury ordinarily must be severe enough to cause loss of consciousness. However, a relatively moderate blow, particularly when delivered to the occiput, can create a permanent perceptive hearing loss.#

The hearing loss is severest for the high frequencies, and the peak loss is usually at 4000 cps. The vertiginous attacks are of the postural type, as described for patients having longitudinal fracture.

The animal experiments show that the deafness is due to injury of the organ of Corti, identical with that which results from a shock pulse in air, as a bomb blast or pistol shot. In later paragraphs we shall develop more fully the concept that a head blow creates a bone-conducted pressure or sound wave which injures the cochlea. We will suggest, furthermore, that postural vertigo is probably the result of injury to the vestibular labyrinth, particularly the otolithic structures of the utricle and saccule, as the result of intense linear acceleration of the head.

References 17-22. Hunter, R. J., in discussion on Lindsay,[23] p. 175.

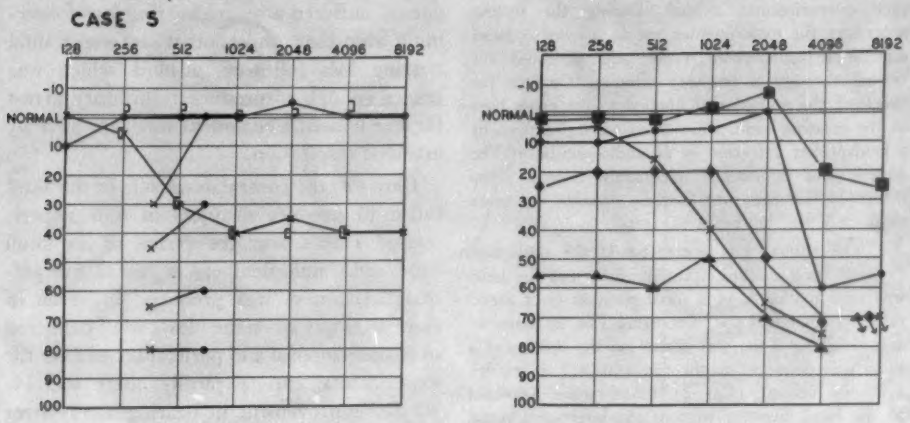


Fig. 3.—Labyrinthine concussion: *Case 5*, audiogram made 48 hours after head injury without temporal-bone fracture. There is a perceptive type of deafness for frequencies from 512 to 8192 cps, and loudness recruitment is complete for the 512 cps frequency. See text (page 522) for explanation of significance of recruitment. The chart on the right shows the air-conduction thresholds for each of five patients in whom there was temporal-bone fracture in the opposite ear. The greatest losses are for high frequencies, particularly the 4096 cps.

The following case is representative of this type:

CASE 5.—A 21-year-old steel worker fell from an automobile, striking his head on the pavement. He was unconscious for one hour. There was no bleeding from his ears. Roentgenograms revealed an occipital skull fracture but no evidence of temporal-bone fracture. Lumbar spinal tap the next day showed grossly bloody spinal fluid. He complained of hearing loss in the left ear and of dizziness on postural movement. He had considered his hearing to be normal prior to injury. Examination 48 hours after injury revealed normal eardrums. Audiometric study showed a perceptive deafness in the left ear for frequencies of 512 cps and above (Fig. 3, Case 5). The Weber test, performed on the audiometer, revealed lateralization to the right ear for all frequencies from 512 to 4096 cps. Loudness recruitment was present for the 512 cps frequency.

The significance of loudness recruitment is discussed later in this paper. Suffice it to say at this time that loudness recruitment is characteristically present in perceptive deafness resulting from a blow to the head, and its presence strongly supports the concept that damage is to the sense organ.[37]

Hearing losses occurring in five patients in the ear opposite a temporal-bone fracture are illustrated in the right chart in Figure 3.

II. EXPERIMENTAL FINDINGS

The following experiment was performed to provide further information on the pathological changes in the auditory and vestibular systems resulting from blows to the head. Some of the results have been published previously.[38] Ten cats were used, and the experimental procedure for each was as follows:

1. The function of one ear was destroyed in a sterile operation by macerating the cochlea with a probe.

2. When the animal had recovered from the acute vestibular reaction following this procedure he was trained to respond to pure-tone auditory stimuli by walking forward in a rotating cage.* An easily audible 1000 cps tone was presented for three seconds, followed by electric shock and buzzer of one-second duration. When the animal had learned to respond to this tone by forward movement, the shock was omitted, except for occasional reinforcement. Once the animal had learned the response, it was easy to train it to respond to the series of test frequencies (125 to 16,000 cps at octave intervals). The threshold for each test frequency was determined by attenuating the intensity of the stimulus tone in 5 db. steps during repeated tests to a point where correct responses were made only 50% of the time. By this procedure an audiogram was obtained for

* References 4 and 27.

each exeperimental animal. During the testing procedure the experimenter sat at a control panel outside a sound-proof room and observed the animal through a one-way window. Current for shocking the animal was applied to the cross bars of the rotating cage. Pure tones were produced by a loudspeaker activated by an audio-oscillator. The animal was considered adequately trained when thresholds for each test frequency varied not more than ± 5 db. on repeated tests.

3. The animal was anesthetized; the skull was exposed by a midline scalp incision, and the head was held manually in a fixed position on a metal table top. A metal rod, measuring 1 in. in diameter and weighing 2 lb., was placed on the skull, and a blow was delivered to the rod with a 1 lb. mallet. For one animal (Cat 2) blows were delivered to the head directly, without the interposed metal rod. The scalp incision was closed with nonabsorable surgical (silk) sutures. In no case was there bleeding from the ears. In eight instances the blows failed to produce hearing loss and the procedure was repeated after one or two weeks. Anesthesia for all procedures consisted of pentobarbital (Nembutal) administered intraperitoneally and supplemented by intravenous thiopental (Pentothal) sodium. Penicillin was given postoperatively for five days.

4. Auditory testing was resumed two days later, and thresholds were again determined for all test frequencies. The animals were tested once or twice a week until killed.

5. At periods of time, varying from two days to five months, the animals were killed by perfusion with isotonic saline solution, followed by 20% formalin solution. The brains and temporal bones were removed and prepared for histological examination. The temporal bones were sectioned at 20μ, and every fifth section was stained and mounted. Each cochlea was graphically reconstructed. By this method it is possible to give lesions of the organ of Corti and spiral ganglion a spatial orientation within the cochlea. The method has been described in detail elsewhere.† Nissl stains were made from selected regions of the auditory cortex and medulla of Cats 4, 5, and 10.

In order to produce hearing losses rather consistently, the blows to the skull had to be severe enough to produce temporary respiratory arrest, pupillary dilation, and loss of corneal reflex. Usually the degree of deafness varied directly with the severity of the cerebral concussion produced by the head blow. There were exceptions, however, for one cat suffered a severe hearing loss following a mild blow and another suffered a mild hearing loss following a blow which was severe enough to produce respiratory arrest for five hours. The animal was kept alive by artificial respiration.

Blows to the contralateral side of the skull failed to produce deafness in four experiments. Blows over the vertex of the skull produced a mild deafness in two of five animals. Deafness was produced in seven of eight attempts when the blows were delivered over the temporal and parietal bones near the experimental ear. Typically, there was 14-40 db. improvement in hearing acuity over the frequency range tested during the first two weeks following the head blow (Fig. 4). For 24-48 hours following recovery from the anesthetic the animals were sluggish, disliked being disturbed, and seemed to be unsteady, although it was difficult to determine whether this was a true vestibular ataxia or the residual effect of pentobarbital anesthesia. None of the animals had spontaneous nystagmus following the head blows. Vestibular function tests and postural vertigo tests were not performed on the animals.

Four of the ten cats had skull fractures, which were confirmed on gross examination at autopsy, but the temporal bone was involved in only one cat. In this animal a small linear streak involved the bony wall of the posterior semicircular canal but there was no evidence of injury to the membranous labyrinth on histological examination. Cat 5 received an intravenous injection of 10 cc. of 1% solution of trypan blue 18 hours and, again, 42 hours after injury. The animal was killed 48 hours after injury, and gross examination of the brain revealed staining of the superficial layers of the cortex, as seen in Figure 5*A*. Deeper areas of the cerebrum, as well as the cerebellum and medulla, were free of stain. This cat suffered a moderately severe hearing loss. Three brains showed areas of focal cortical degeneration and cyst formation. Cat 2 sustained a mild injury to the midportion of the ectosylvian gyrus (Fig. 5*B*). The auditory cortex of the cat is located

† References 15 and 39.

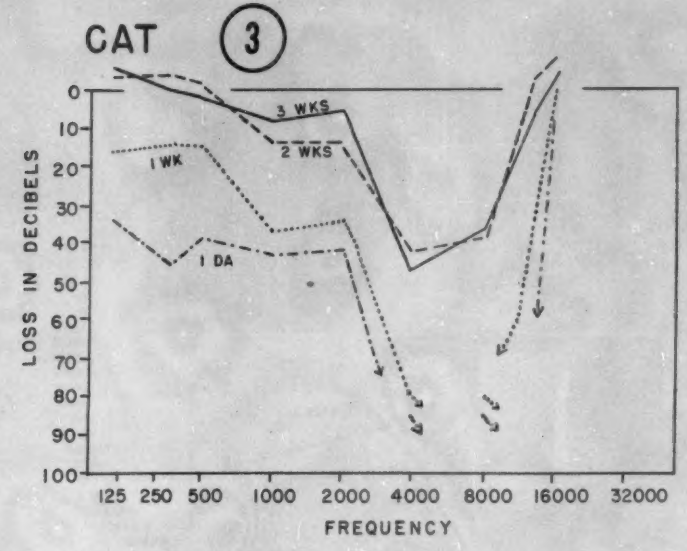


Fig. 4.—Audiograms of Cat 3, showing improvement in auditory thresholds occurring during a two-week interval following an experimental blow to the head. The hearing did not improve after the second week. Cochlear damage is shown in Figure 6.

in the median ectosylvian gyrus, anterior ectosylvian gyrus, posterior ectosylvian sulcus, and pseudosylvian gyrus.[17] It should be pointed out that, even if there had been severe lesions of the auditory pathways or cortex, it is doubtful they would have created threshold losses, for Neff and co-workers have found auditory thresholds in the cat, tested by the same technique as we used, to be unchanged after bilateral ablation of the auditory cortex.‡ Apropos to this subject is another experiment in which we have shown that up to 75% of nerve fibers supplying a particular region of the cochlea can be destroyed without creating threshold losses for frequencies having their locus in that region.§ In none of the ears was there evidence of laceration of the cochlear nerve or bleeding into the internal auditory meatus.

Cats 1 and 2, killed one month after injury, had very slight threshold losses for the 4000 cps frequency, with no evidence of cochlear injury. We suspect that the changes in the sense organ of these ears were too subtle to detect with existing histological techniques. Cats 3, 4, 5, and 6 had hearing losses restricted to the 2000 to 8000 cps range and cochlear lesions restricted to the upper basal and lower middle turns. Cats 7 and 8 had severe hearing losses involving a wide range of frequencies and severe, widespread cochlear injury. Cat 9 did not respond to the tones at maximum intensities after injury (90 db. above preinjury threshold in the range 100 to 2000 cps), and histological examination revealed a complete loss of hair cells throughout the entire cochlea. No postconcussion audiogram was obtained in Cat 10 because brain damage had produced a partial loss of motor function and behavioral tests could not be made.

The findings in three representative ears are shown in charts in Figure 6. In Figure 6*A* the graphic reconstruction and audiogram for Cat 3 are shown. A similar spiral graphic reconstruction was made for each ear, and the information was transferred to linear barograms, as shown in Figure 6 *B*. The audiogram and cochlear chart for each ear are displayed so that the frequency scale of the audiogram and the distance scale of the cochlear chart are on parallel and equal-length coordinates. The frequencies are located on

‡ Neff, W. D., and others: Personal communication to the authors (unpublished work, Department of Physiological Psychology, University of Chicago).

§ References 40 and 41.

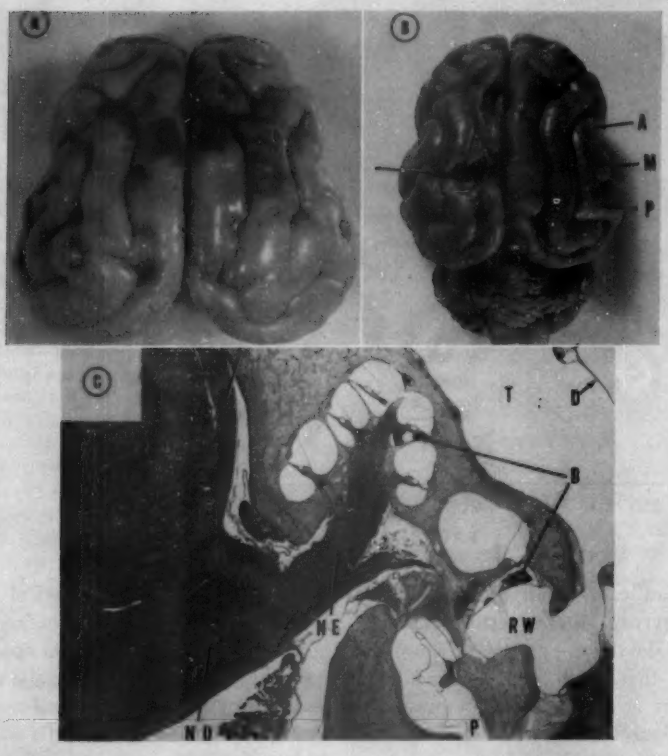


Fig. 5.—*A*, cortical staining with trypan blue 48 hours after blow to vertex of skull. Both auditory cortices are normal.

B (Cat 2), areas of focal cortical degeneration and cyst formation after a blow to left parietal region. Note slight injury to left middle ectosylvian gyrus (arrow). The lesion would not cause an auditory threshold elevation (for explanation see text, page 519). For orientation, the anterior (*A*), middle (*M*), and posterior (*P*) ectosylvian gyri, which constitute the primary auditory areas of the cat, are indicated on the opposite side.

C, photomicrograph from serial sections showing the medulla and cochlea. *NU*, cochlear nucleus; *NE*, cochlear nerve; *B*, blood in scala tympani of basal and apical turns; *RW*, round window; *P*, posterior semicircular canal; *V*, trigeminal nerve; *T*, tympanic cavity; *D*, eardrum.

the abscissa of the audiogram in accordance with the anatomical frequency scale, which takes into account the spatial distribution of frequency along the organ of Corti. The data upon which the anatomical frequency scale for the cat is based have been published elsewhere.[30] Hearing losses are shown in decibels relative to the animal's preinjury threshold. The black filling in the cochlear charts indicates the percentage of elements missing. "First order neurons" refers to the spiral ganglion cells and their fibers. The charts show that Cat 3, with a moderately severe hearing loss for the 4000 and 8000 cps frequencies, had a loss of outer hair cells in the region between 7.75 and 10.5 mm. Cat 4 had a hearing loss involving the range between 2000 and 12,000 cps and hair-cell damage extending from 6 to 15 mm. Cat 7, with a profound deafness for all frequencies above 500 cps, had damage to the organ of Corti throughout the basal 18 mm. of the cochlea. Frequencies below 500 cps were not tested in this animal.

In regard to the reliability of the behavioral audiogram, the question is sometimes raised as to whether loud tones, particularly low tones, might not create vibrations which could

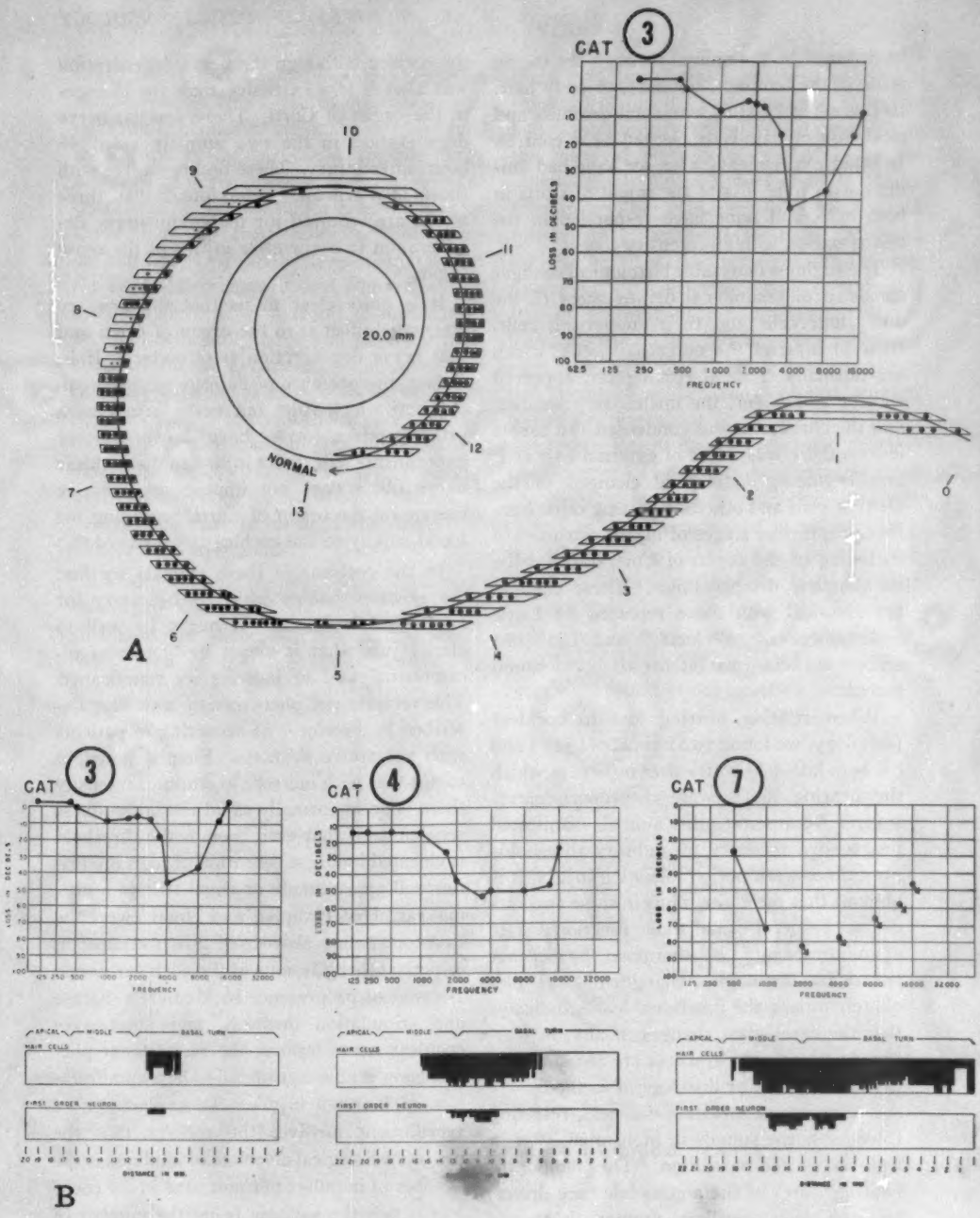


Fig. 6.—*A* (Cat 3), cochlear reconstruction and audiogram. Graphic reconstructions are made for the ear from serial microscopic sections of that ear so that the pathological changes can be spacially oriented within the cochlea. The quadrilateral boxes represent organs of Corti, and the black dots represent hair cells. Distance is indicated in millimeters. A loss of outer hair cells from 7.75 to 10.5 mm. resulted in a threshold loss for the frequencies 4000 and 8000 cps. See chart in *B* for further graphic representation of this relationship of hair-cell injury to hearing loss.

B, audiograms and cochlear charts for Cats 3, 4, and 7, showing relationship of cochlear injury to hearing loss for each animal. The arrows in the audiogram for Cat 7 indicate that those frequencies were not heard at maximum available intensity. See text (page 519) for detailed description of charting method.

be detected by the animal through the tactile sense of the footpads, fibrissae, or body hair. In this regard Cat 9, with total deafness and total loss of hair cells, served as a control. In other experiments, also, we have had animals with total loss of the organ of Corti in both ears, and none have responded in the test situation to high-intensity tones.

The slightest detectable histological changes consisted of anatomical derangement of the outer hair cells and their supporting cells. In mild injuries the outer hair cells, which are normally tall and rectangular, appeared shorter and wider, the nuclei were smaller, and the chromatin was condensed. In severe lesions there was a loss of external hair cells and beginning cytological changes in the Deiters' cells and other supporting cells. Further progressive stages of injury consisted of flattening of the organ of Corti and, finally, its complete disappearance. These changes are identical with those reported by Lurie and associates,[24] Perlman,[31] and Lindquist and co-workers [22] for intense air-borne sound stimuli.

When relating hearing loss to cochlear pathology, we found two animals (Cats 5 and 8), both killed 48 hours after injury, in which the hearing losses were disproportionately severe. Because the other animals manifested progressive recovery of auditory thresholds for a three-week period following injury, it is obvious that the audiograms in these two cats did not reflect the final stable functional state of auditory acuity. Furthermore, the fact that the visible histological changes did not adequately mirror the functional losses indicates that the cytological changes, if any, in temporary stimulation deafness are too subtle to detect with routine histological methods.

Temporary stimulation deafness, resulting from air-borne sounds of high intensity, is a well-recognized condition. The temporary hearing losses of the automobile race driver, airplane pilot, artillery gunner, industrial laborer, etc., are well known and referred to as the "threshold shift." [5]

The six animals which were killed three weeks or longer after injury had partial loss of the nerve supply to the damaged region of the cochlea, although the nerve degeneration was always less extensive than the changes in the organ of Corti. There was no nerve degeneration in the two animals killed 48 hours after injury. These findings agree with those of Perlman,[31] who found that three weeks are required for maximum nerve degeneration to occur after injury of the sense organ.

It is quite clear to us that the primary traumatic effect is to the organ of Corti and that nerve degeneration is secondary. It is, in fact, the presence of damage to the organ of Corti which rules out nerve injury as a primary effect, for we have shown in other experiments that direct injury to the cochlear nerve fibers does not impose degenerative change on the organ of Corti, providing the blood supply to the cochlea is preserved.[40]

In the cochleae of these animals we find the exact pathology which is necessary for loudness recruitment. It might be well to clarify just what is meant by "loudness recruitment" and to indicate its significance. The recruitment phenomenon was first described by Fowler [12] as occurring in patients with perceptive deafness. Simply stated, it means that with increase in stimulus intensity there is an abnormally rapid rise in loudness sensation, so that even though the threshold is elevated loudness sensation at high intensities will approximate or equal that of a normal ear. Dix, Hallpike, and Hood [7] were the first to note the absence of this phenomenon in patients with lesions of the cochlear nerve. Because of its presence in Ménière's disease and stimulation deafness, and absence in cochlear nerve lesions, the recruitment phenomenon is now considered to be a manifestation of hair-cell injury. An explanation of recruitment involves the concept that the neurophysiological correlate of loudness is the number of impulses per unit time in the cochlear nerve, the variable being the number of nerve fibers involved and the frequency of the impulses in these fibers. Now, if at high stimulus intensities the hair cells which remain in an injured region of the cochlea are capable of exciting a normal complement of

nerve fibers, then normal loudness sensation should result. This is truly a hypothesis and does not explain how residual inner hair cells in a pathological ear could activate large numbers of nerve fibers at high stimulus intensities. It is known that a great number of nerve fibers have their endings only on external hair cells. One possible explanation is that the electrical excitatory field which develops around hair cells as a result of stimulation becomes sufficiently large and strong at high stimulus intensities that remaining inner hair cells are capable of activating nerve fibers lying in their vicinity, although they would normally be activated by the now missing outer hair cells. The pathological changes which occurred in the vestibular systems of these animals are included in the discussion of positional nystagmus.

Blood was found in the cochleae of five cats. Individual red blood corpuscles appeared normal in size, shape, and staining characteristics, even in the one animal killed five months following injury. In no case was there evidence of tissue reaction due to the presence of blood. In one animal killed 48 hours after injury there were leucocytes in the mass of erythrocytes, whereas in the other animals, killed three weeks or longer after injury, no leucocytes were present. In subsequent experiments we have confirmed the fact that fresh intracochlear hemorrhages contain leucocytes and old ones do not. It is of interest that in each cochlea the erythrocytes had collected to adjacent regions of each turn (Fig. 7). The evidence suggests the erythrocytes behaved as inert foreign bodies floating about in the cochlear fluids and responding to fluid currents and gravitational forces. The sites of hemorrhages were not evident, but we assume that they occurred in small arteries or veins supplying the membranous labyrinth. Although the blood could have reached the scala tympani through the periotic duct from the subarachnoid space in some cochleae, this could not have happened in Cat 10, in which erythrocytes were found in the intact cochlear duct.

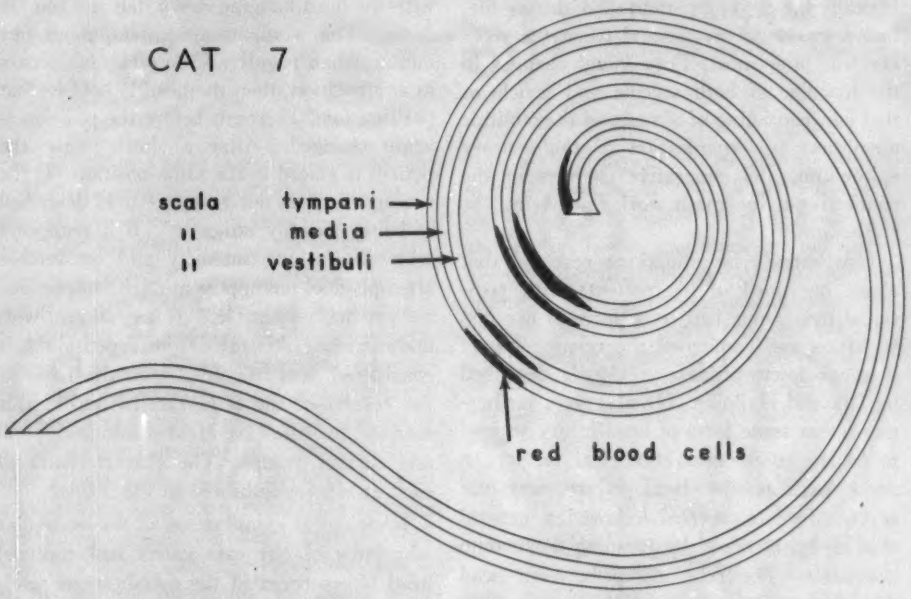


Fig. 7.—Three-channel spiral representing scala vestibuli, media, and tympani developed from the graphic reconstruction of the ear of Cat 7. The black filling shows the location of red blood cells within the scalae. The cat was killed six weeks after a blow to the head. The audiogram and cochlear pathology are shown in Figure 6.

III. POSITIONAL NYSTAGMUS IN HEAD INJURY

Positional nystagmus is a self-limiting benign disorder which commonly follows head injury but may also occur spontaneously in patients with no history of head injury. Bárány, who first reported on this disorder in 1921,[1] described two types, attributing one type to disorder of the static labyrinth and the other to lesions of the central nervous system. Subsequently, Nylen,‖ Lindsay,[28] Dix and Hallpike,[8] Cawthorne,[3] Hunter,¶ and others have reported further on this interesting condition.

Several recent investigations support the concept that positional nystagmus may be caused by disorders of the vestibular labyrinth. Dix and Hallpike [8] found that about two-thirds of 100 patients with positional nystagmus had evidence of ear disease. They noted, furthermore, that in most patients in whom the ear disease was unilateral the positional nystagmus occurred when that ear was placed undermost during postural tests. They also had the opportunity of examining histologically the ear of a patient who, during life, had a positional nystagmus when the right ear was undermost. They found changes in the maculae of both saccule and utricle of that ear, consisting of absence of the otolithic membrane, disorganization of the sensory epithelium, and generative changes in the connective-tissue mesh work underlying the epithelium.

Cawthorne's investigations revealed that about one-third of his patients with positional nystagmus had some form of ear disturbance and confirmed the relationship of it to ear-downwardness previously described by Dix and Hallpike. He observed, furthermore, that some form of head injury seemed to be the commonest etiological factor. In some patients the head injury was not severe; in fact, several followed a general shaking-up incurred by jumping down onto the heels. The relationship between head injury and positional nystagmus has been noted by others.[18]

It has now become routine at our clinic to do positional nystagmus tests on all patients complaining of paroxysmal vertigo. We consider these postural tests to be a simple, effective way of inquiring into the integrity of the static labyrinth.

We perform the test after the method of Cawthorne as follows: The patient is seated on a couch or examining table and told to keep the eyes open and look straight forward, even though he might feel dizzy or giddy. The examiner holds the patient's head between his hands and lays him back on the couch in a supine position with the head to one side, a movement requiring about three seconds. If there is no nystagmus after 10 seconds the patient again is brought to the sitting position and examined in this position for 10 seconds. He is then laid back in the supine position with his head to the other side. After 10 seconds he is brought upright again and observed for nystagmus. Finally, he is placed back again, this time with the head hanging down, but not too far down. The whole test occupies about one minute when negative. If nystagmus occurs in any position, then the head is held in that position for 30 seconds before the position is again changed. After a short pause the patient is placed in the same position. If the nystagmus does not reappear it is described as being "readily fatigable." If it reappears with diminishing intensity and on further attempts does not appear at all, it is described as "gradually fatigable." If it continues with undiminished intensity despite repetition, it is considered "nonfatigable." On the basis of the results of the tests carried out in this manner, the cases are divided into peripheral and central groups. The characteristics of each group are indicated in the Table.

Histological examination of the vestibular labyrinths of our cats which had received head blows revealed the membranous semicircular canals and the cristi ampullari to be normal in all animals. The static labyrinths (utricle and saccule) were normal in all but

‖ References 28 and 29.

¶ Hunter, R. J., in discussion on Lindsay,[28] p. 175.

Cat 2. In this animal there was a tear in the membranous walls of both utricle and saccule. The wall of the saccule had collapsed and lay on the otolithic membrane, compressing it against the saccular macula (Fig. 8). There was about 30% decrease in hair-cell population in the saccular macula. The utricular macula was normal. The method used to produce injury in this animal differed somewhat from that of the other animals. In addition to receiving several blows to the head by a hammer hitting a heavy brass rod placed against the skull, it also received two blows directly to the skull with the hammer. These latter blows were delivered to the exposed skull with the animal lying on its side so that the head was allowed to accelerate freely. When first tested, 24 hours after injury, this animal had a 45 db. hearing loss for 4000 cps, and at the time it was killed, at six weeks,—had a loss of 20 db. for 4000 cps.

Although the function of the saccule has not been definitely determined, the utricle is known to respond to forces of linear acceleration and to gravity, thus providing information on linear movements and positional changes of the head. This is accomplished by a mechanism consisting of a sensory structure (the macula) upon which is mounted a gelatinous mass (the otolithic membrane) containing calcium carbonate crystals (calcite, S.G. 2.71). These crystals lend weight to the otolithic membrane, causing it to be displaced by the forces of linear acceleration and gravity. We believe, therefore, that the static labyrinth (utricle and saccule), which is mechanically designed to detect fine changes in linear acceleration, may be injured by the intense stimulation it receives from a head blow. Therefore, like the cochlear injury already described, it may be referred to as a "stimulation" injury.

Windle, Groat, and Fox [46] have shown that degenerative changes in the lateral vestibular nuclei also may result from experimental blows to the head. We cannot confirm or deny this finding, as we did not study the vestibular nuclei in our animals.

IV. SUMMARY AND CONCLUSIONS

In our experience, partial permanent deafness occurs in about 50% of patients who incur a blow to the head severe enough to produce unconsciousness. Even a mild head blow without loss of consciousness can occasionally result in deafness. Usually the hearing loss involves a wide range of frequencies and is of sufficient magnitude for the patient to be aware of it. However, a patient sustaining a high-frequency hearing loss, particularly when it involves only one ear, may not be aware of it, and in such cases only audiometric testing will reveal it. Deafness from blows to the head may be classified into three groups.

Characteristics of Cases Belonging to the Peripheral and Central Groups

	Peripheral Group	Central Group
Direction of nystagmus	Constant in different head positions	May vary with different head positions
Onset of nystagmus after assuming new posture	Latency period of 3 to 10 seconds	Immediate onset
Amplitude of nystagmus	Usually large	Usually small
Duration of nystagmus	3 to 10 seconds	Much longer, may continue indefinitely so long as provocative posture is maintained
Fatigability of response to repetition of posture change	Nystagmus cannot be elicited more than 1, 2, or 3 times in diminishing intensity	Nystagmus recurs each time provocative posture is assumed
Sensation of vertigo	Severe	Mild
Associated nausea	Common	Rare

A. Longitudinal Fracture of the Temporal Bone

Longitudinal fracture of the temporal bone extends through the middle ear and mastoid bone and results in conduction deafness through interference with the function of the sound-conducting apparatus, both by direct injury and by imbedding them in blood.

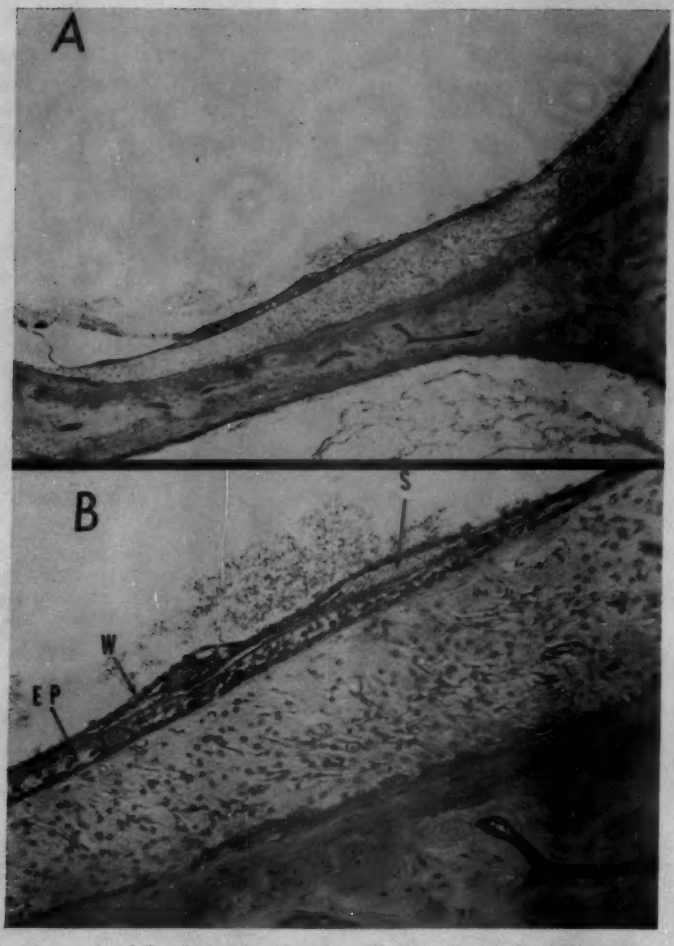


Fig. 8.—*A*, saccule of Cat 2, showing that the wall of the saccule has collapsed, compressing the atrophied otolithic membrane against the macula. *B*, high-power view of saccule to show collapsed saccular wall (*W*), which has trapped a group of otoconia (stataconia) (*S*). There is about a 30% decrease in hair cells in the epithelial layer (*EP*).

There often exists, in addition, a perceptive deafness for high frequencies as the result of stimulation injury to the organ of Corti. Longitudinal fracture, therefore, is characterized by rupture of the tympanic membrane and bleeding from the ear and an associated deafness which may be either purely conductive or of the combined perceptive-conductive type. The conductive deafness is usually totally reversible in three to six weeks. Recovery of the perceptive loss is almost never complete, although some recovery can be expected during the first three weeks after injury.

B. Transverse Fracture of the Temporal Bone

Fracture of the temporal bone may occur transverse to the long axis of the petrous bone passing through the labyrinth, thereby creating a profound loss of both auditory and vestibular function. Clinically there is a bluish discoloration of the eardrum due to a blood-filled middle ear, profound hearing loss, occasional facial palsy, and intense vertigo with

nystagmus directed to the normal ear. The vertigo gradually subsides over a three- to eight-week period. Deafness is permanent.

C. Labyrinthine Concussion

Perceptive deafness may occur in an ear in which there is no clinical or roentgenological evidence of temporal-bone fracture. This loss is greatest for the high frequencies, particularly for the 4000 cps frequency, and in this regard is identical with deafness resulting from intense air-borne sounds. Some recovery of hearing can be expected during the first three weeks following injury.

To determine the pathology responsible for the perceptive deafness and vertigo an experiment was performed in which head blows were delivered to 10 conditioned cats. Hearing tests and histological studies were made of the brains and inner ears. The hearing losses were identical with those occurring in human patients, the characteristic feature being a maximum loss at the 4000 cps frequency. Histological examination of the auditory system revealed the significant pathological changes to lie in the cochlea, where there was damage to the organ of Corti, severest in the upper basal turn, the region serving the 4000 cps frequency. These changes are identical with those which have been produced experimentally by air-borne blasts and noises of high intensity. Thus, a blow to the head, like a blast in air, appears to create such violent displacement of the basilar membrane and organ of Corti that both reversible and irreversible cellular injuries result.

D. Positional Nystagmus

Positional nystagmus may persist for several months following a head blow; it may follow an only mild jarring of the head. It may occur independently of deafness. Positional nystagmus is a disorder characterized by transient attacks of vertigo and nystagmus when certain head postures are assumed. It is best elicited by moving the patient from a sitting to a supine position with the head to the side so that the injured ear is downmost. The underlying pathological change responsible for the positional nystagmus is thought to be injury to the utricle or saccule. We believe that these structures, which are sensitive to linear acceleration and deceleration, are injured by the intense stimulation of a head blow. In 1 of 10 cats the head blows were delivered so as to allow free acceleration of the head. The experimental ear of this animal incurred a rupture of the membranous walls of the utricle and saccule and degenerative changes in the macula of the saccule.

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Case Reports

FACIAL PARALYSIS AND MUSCLE-RELAXANT ANESTHESIA

PAUL M. OSMUN, M.D., Seattle

THE FOLLOWING case report is presented primarily to forewarn otologists of the insidious danger of using succinylcholine-drip muscle-relaxant anesthesia in any surgical procedure where the identification of the seventh cranial nerve is important to its preservation.

REPORT OF CASE

A 32-year-old woman had chronic left mastoiditis of 10 years' duration. The history and findings follow: severe headache for one month, dizziness and nausea for two weeks associated with a positive fistula test.

A radical mastoidectomy was performed on Nov. 7th, 1955. At operation, the mastoid bone was found to be eburnated. No cells were encountered; the posterior auditory canal wall and the adjoining mastoid area were taken down to the bridge simultaneously by means of gouges and mallet. As the bridge was being thinned down, what appeared to be a mastoid cell was uncovered 1 mm. below the horizontal semicircular canal and 1 mm. behind the crest of the facial ridge.

Despite the superficiality of its location, the cell or nerve was pressed with the back of a curette while the anesthesiologist watched the patient's face. There was no muscle response. However, this area was avoided, the bridge taken down, and the mastoid operation was completed. The middle ear was a mass of necrotic tissue, all bound down as a solid piece in which only fragments of the ossicles were identifiable, the remainder having succumbed to a long-standing process of osteomyelitis.

Because of the relative elevation of the facial ridge and the need to lower it, the aforementioned mastoid cell or nerve was uncovered to a length of approximately 2 mm. In the course of lowering the facial ridge anterior to the suspicion-arousing area, the nerve or mastoid cell was stimulated with the back of a curette on four occasions while the anesthesiologist observed the face. There was no response.

The wound was closed, and the cavity was lightly filled with tetracycline (Achromycin)-hydrocortisone half-inch tape packing.

At this time, the anesthetic was discontinued and the patient immediately regained consciousness. There was a total paralysis of the face on the side operated on.

The rapid recovery of the patient led to an inquiry as to the type of anesthesia that had been given. The patient had been given a continuous drip of succinylcholine, 1 mm/cc., plus nitrous oxide and oxygen by intubation. At this time, it occurred to the operator that when the anesthesiologist had been asked to look at the face he was very anxious to get back to the rebreathing bag because respirations were dependent upon manipulation of the bag.

In response to additional inquiry, the anesthesiologist said that succinylcholine acted upon the myroneural junction and had been given in such quantity as to cause complete muscle relaxation. This meant that stimulation by pressure on the facial nerve in order to elicit a response in the face for the purpose of nerve identification was not a reliable procedure.

The following day the cavity was reexplored and the suspicion-arousing area aforementioned was uncovered in its descending portion for a distance of 1.5 cm. There was no response in the face with use of a non-muscle-relaxant anesthesia. The facial nerve proved to be grossly swollen and injected. A small black linear streak was observed in the horizontal canal; pressure against the area of the stapes caused bubbles to come out of the fistula.

Accepted for publication Jan. 13, 1956.

1116 Summit Ave. (1).

Abstracts from Current Literature

Ear

FENESTRATION—PAST AND FUTURE. GEORGE E. SHAMBAUGH JR., Ann. Otol. Rhin. & Laryng. **64**:149 (March) 1955.

Shambaugh reviews the past history of the fenestration operation and comments on its present status.

Future development in fenestration surgery will probably include improved methods for preventing postoperative serous labyrinthitis, techniques for overcoming the residual conductive loss remaining after successful fenestration, and more definitive information regarding the permanence of the restored hearing.

Today's most frequent cause of failure appears to be postoperative serous labyrinthitis. Studies of the experimental animal indicate hemorrhage into the labyrinth and infection of the cavity are contributing factors. Utmost care is necessary to maintain aseptic technique during the operation and throughout the first month of postoperative treatment. With routine antibiotic therapy infection of the wound is virtually never seen, and healing by epidermization occurs without granulations or suppuration. Nonetheless, serous labyrinthitis still causes about 5% of operations to fail, perhaps due to factors not yet known.

Blocking the tympanic membrane in the fenestrated ear to enhance the sound-pressure differential between the fenestra and the round window holds promise of future reductions in the residual conductive loss after fenestration.

It is illogical to expect fenestration to retard cochlear nerve deterioration in cases of otosclerosis, yet there is some preliminary evidence suggesting that in certain cases better cochlear nerve function is maintained in the fenestrated ear than in the ear not operated on. Future long-term study should confirm or disprove this fact.

Speculation regarding the future of fenestration versus stapes mobilization must remain as pure speculation until more is known of the latter procedure. It is conceivable that the simpler mobilization may prove to be an optional preliminary procedure for certain cases of incomplete stapes ankylosis. Considering the solid mass of otosclerotic bone that frequently replaces the stapedial footplate and oval window, stapes mobilization cannot conceivably replace fenestration in cases of advanced stapes ankylosis. STEELE, Kansas City, Kan.

THE PATHOLOGY OF ACUTE DIFFUSE EXTERNAL OTITIS. L. H. SOPHIAN and B. H. SENTURIA, Ann. Otol. Rhin. & Laryng. **64**:300 (March) 1955.

The authors studied biopsy specimens taken from the skin of five patients presenting the clinical symptoms and signs of acute diffuse external otitis.

All biopsy specimens showed evidence of hyperkeratosis, edema, acanthosis, parakeratosis, and hyperplasia of the epidermis. The capillary and arteriolar vessels showed dilatation, endothelial swelling, hyperplasia, and occasional thrombosis and necrosis. Such vascular reactions are usually seen in toxic and allergic inflammations but rarely in purely pyogenic infections. The exudate showed a high content of the eosinophiles, which again differs from the type of infiltrate in suppurative inflammations. Therefore, Sophian and Senturia state that there seems to be certain clinical and pathologic aspects of the picture which are compatible with a state of sensitization.

The apocrine glands showed a leucocytic infiltration and inflammation. Compression of these glands was apparently due to edema and infiltration of the periglandular fibrous tissue. Degeneration of the glandular epithelium may have resulted from interference with the vascular supply. The sebaceous glands were involved to a slighter extent, but no conspicuous degenerative changes were observed.

A number of photomicrographs are presented. STEELE, Kansas City, Kan.

SOME CLINICAL IMPLICATIONS OF RECENT EXPERIMENTS ON THE PSYCHOLOGY OF HEARING. D. E. BROADBENT, Proc. Roy. Soc. Med. **48**:961 (Nov.) 1955.

The occasion was a joint meeting of the Section of Otology with the Section of Laryngology held at the Anatomy Theatre, Downing St., Cambridge, England, on June 16, 1955, with Josephine Collier, F.R.C.S., presiding. Mr. Broadbent, the speaker, works at the Allied Psychology Research Unit, Cambridge. He said that hearing aids are usually, but not always, monaural affairs operating from a single microphone. How can we help the novice who comes for hearing tests? Since hearing is a function of the brain, there are some persons who hear sounds which they cannot interpret. This is particularly true when the sounds are unusual, as in a foreign language, where, although the sounds may be loud enough, they are meaningless because they have not been heard before or, at least, are not understood. Thus there is an increased effort to hear. There is the matter of listening to two things at once and trying to unscramble them and place them in orderly sequence. Experiments are described in the text to illustrate this phenomenon. It may be that one cerebral hemisphere can inhibit the other! People more frequently do a "double-take" in hearing than in vision. Again, people can learn to judge the direction of sound from its quality with a fair degree of accuracy, and a difference in pitch helps in dealing with two simultaneous sounds. Auditory fatigue is a well-recognized item in testing. Reference is made to the experiments of Rawdon-Smith (1936), who found that one could "disinhibit" auditory fatigue. The conclusion is that noise which produces deafness has an effect in the cochlea rather than in the central nervous system. There are, however, some who object to this opinion. In any case, one must beware in taking audiograms not to allow the factor of fatigue to enter into the ultimate data.

Finally, there are two points which Broadbent set out to make: "Binaural hearing is tremendously important in complex auditory situations; the audiogram may sometimes be unduly discouraging if the patient has heard too long, too monotonous, too unvaried a series of tests."

I. W. VOORHEES, New York.

THE DISABILITY OF CHRONIC EAR DISEASE IN RELATION TO INSURANCE AND EMPLOYMENT. A. BOWEN-DAVIES, Proc. Roy. Soc. Med. **48**:968 (Nov.) 1955.

This is a study of otology in relation to employment, with special reference to suppurative otitis. The point is made that in these days fatalities due to chronic ear disease are rare, with or without radical mastoidectomy. Consultation with medical divisions of insurance companies revealed the opinion that "little account should be taken of this condition when assessing for life insurance." Most cases with running ears are now accepted at standard rates. The size and site of perforations are stressed; a small attic perforation is "least favored" and justifies a small increase in premium. Persons with large dry perforations are accepted, but those with fetid perforations are rejected until treated. Deafness is not ordinarily a cause for rejection, but Ménière's disease is regarded as a serious condition. The Armed Services have always insisted that the men have normal drums with at least satisfactory hearing. A soldier must be able to serve in any and all climates; therefore, a diseased ear is cause for rejection. Moreover, one with poor hearing might be shot by a sentry if he failed to halt! The General Post Office is fairly lenient in considering applicants with aural disabilities and uses common sense in placing them. However, severe deafness is a liability to a postman, who must travel in traffic. Where no adequate medical service is possible to a patient with chronic purulent ears, he should be taught how to cleanse the canal and to instill medication.

I. W. VOORHEES, New York.

PURE-TONE AUDIOMETRY FOR PRESCHOOL CHILDREN. BENGT BARR, Nord. med. **51**:464 (April 1) 1954.

The author has performed pure-tone audiometric investigations on about 300 children 1-6 years of age. Both psychogalvanic skin-resistance (PGSR) audiometry and play audiometry were used. The procedures followed and the results obtained with the respective methods are discussed and compared. It was possible to use play audiometry on normally developed children as young as 2½ to 3 years of age. PGSR audiometry could be used on even younger children. The threshold values obtained were considered reliable when two audiograms showed agreement; assuming a difference of not more than 5 db. as permissable, the two methods showed full

agreement in 85% of the threshold determinations. Follow-up examinations performed one to two years later showed practically unchanged values. The author is of the opinion that in cases where play audiometry can be carried out it should be the method of choice.

WALLGREN, Stockholm [A. M. A. J. DIS. CHILD.].

Pharynx

THE CRICOPHARYNGEUS MUSCLE. OSCAR V. BATSON, Ann. Otol. Rhin. & Laryng. **64**:47 (March) 1955.

The cricopharyngeus muscle, first described in 1717 by Valsalva as an anatomic entity, has been studied by Batson in the cadaver and has been experimentally examined in the freshly killed dog in a state of rigor. The existence of the horizontal bundle of muscle fibers, which Valsalva named on a morphological basis, has been established by direct observation and cinefluorography in the living and, in the contracted state, by an anatomic demonstration in the cadaver. Therefore, previous clinical observations are confirmed and the cricopharyngeus muscle is established as a definite anatomic physiologic structure.

STEELE, Kansas City, Kan.

Larynx

CARCINOMA OF THE LARYNX AND ITS PRESENT-DAY MANAGEMENT. FREDERICK A. FIGI, Ann. Otol. Rhin. & Laryng. **64**:136 (March) 1955.

Figi maintains that carcinoma of the larynx can be cured in an appreciably higher percentage of cases than at present if the lesions are diagnosed in an earlier stage. The most important diagnostic features should include a detailed history, careful mirror examination, and biopsy.

The therapeutic measures in the treatment of laryngeal cancer include thyrotomy, total laryngectomy, hemilaryngectomy or partial laryngectomy, pharyngotomy, endoscopy, and irradiation. Exploration through thyrotomy (laryngofissure) often is necessary to determine the extent of the malignant process and the best means of removal. Suspension laryngoscopy and electrocoagulation are useful in the treatment of many lesions of the supraglottic portion of the larynx. By means of hemilaryngectomy and immediate skin grafting, some moderately advanced carcinomas with varying degrees of fixation can be cured without disturbing laryngeal function.

STEELE, Kansas City, Kan.

PRACTICAL CONSIDERATIONS OF ANESTHESIA IN BRONCHOESOPHAGOLOGY. CLYDE A. HEATLY, New York J. Med. **56**:367 (Feb. 1) 1956.

Heatly works in the Rochester School of Medicine and Dentistry. He read this paper before the New York State Medical Society in May, 1955. In choosing an anesthetic for direct tube instrumentation certain variables should always be kept in mind: the patient's age, general condition, and temperament, and the hospital's equipment and personnel. The patient's safety is, of course, primary, and so the anesthetic chosen must be influenced by all of the factors mentioned above. Bronchologists often prefer local to general anesthesia, but in children the latter is to be considered for obvious reasons. Heatly uses succinylcholine (Anectine) intravenously with thiopental, preceded by local anesthesia of the pharynx and larynx. There are some contraindications, however, chiefly related to careful administration based on experience. The paper was discussed by Dr. Orrin E. Anderson, who administers dextrose and fluids with elixir phenobarbital sedation for 24 hours preceding the intubation. Locally 10% cocaine "provides the safest anesthesia. . . . The problem of cardiac arrest is ever present . . . the result of adrenalin exhaustion due to overwork of glands during preoperative apprehension."

ABSTRACTER'S COMMENT: Fatalities in direct tube work occur here as elsewhere, and the cause is too often not determinable. If cocaine is used, it should be guarded by adrenalin to prevent deep and immediate absorption. There is scarcely any other field where the value of trained assistants is of greater importance in maintaining positions of the head and in the passing of instruments. There is some danger in the use of morphine as a preliminary sedative because of its depressant action on the respiratory center in the medulla.

I. W. VOORHEES, New York.

DISCUSSION ON THE MODERN INDICATIONS FOR TRACHEOTOMY, WITH SPECIAL REFERENCE TO THE MANAGEMENT OF THOSE CASES REQUIRING ARTIFICIAL RESPIRATION. ALFRED S. H. WALFORD, Proc. Roy. Soc. Med. **48**:947 (Nov.) 1955.

The Section of Laryngology held a joint meeting with the Section of Otology on June 17, 1955, in the Anatomy Theater at Cambridge, with Prof. V. F. Lambert, M.D., Ch.M., F.R.C.S., as chairman. Mr. Walford held that early tracheotomy was highly desirable: "rather be too early than too late." He spoke of the "almost atavistic aversion to performing this operation, which really can be done quickly, safely, and with very little discomfort to the patient if only it is undertaken before he is *in extremis.*" By using a cuffed tube one can seal off the lower airway from the upper and thus protect the lungs from foreign bodies, etc. He referred to the work of Prof. Lassen, of Copenhagen, whose paper, published in 1953, dealt with acute respiratory insufficiency in poliomyelitis. Cough in these cases may be insufficient or absent, and thus there is inability to expel fluid from the lungs. This may be partially overcome by placing the patient in the prone position and using suction. The causal agents may be acute anterior poliomyelitis; other diseases of brain and spinal cord; head injuries; tetanus; injuries to the neck, mouth, pharynx, and larynx. A central cause of laryngeal obstruction may be mistaken for foreign body or local inflammatory disease. In any case if position and suction fail to give a fairly free airway, tracheotomy is the procedure of choice. Vomiting and aspiration may suffocate the patient promptly. General anesthesia is preferred (cyclopropane), but the stomach is pumped out before proceeding further. The British prefer low tracheotomy, but in Copenhagen the high position is preferred. Passing a bronchoscope into a bronchus through the tracheal stoma is easily done and must be carried out if there is air insufficiency. Sticky or gluey secretions necessitate an almost constant and continuous watch of the breathing. We as ENT men must see that moisture is supplied to all "gases" entering the lungs.

A brief recital of the poliomyelitis epidemic in Copenhagen in 1952 is recalled by Walford; at that time nearly 50 cases a day were being admitted to hospitals, which ran out of equipment, space, and personnel to give urgent aid. Forced respiration was maintained by medical students and any other persons available.

The advantages and disadvantages of tracheotomy are set down. One of the disadvantages is that patients who might be saved would be "better off dead," but this is in the realm of the unknowable, since "some of the most severe bulbar cases recover completely." In general, tracheotomy is indicated in those who cannot cough, swallow, or maintain a free lower airway. References are made to the work of Milton V. Davis, of Texas, and Baronofsky and Briggs, of St. Paul. A paragraph is devoted to tracheobronchitis in children, "a very dangerous condition." Crusts must be removed through bronchoscopy and with the use of forceps. One may possibly avoid tracheotomy by using antibiotics, moistening the inspired air, having an oxygen tent at hand, and applying "detergents such as Alevaire or Tryptar to help soften the secretions." Tracheotomy destroys the ability to cough; therefore, if this reflex is still present and effective, opening the trachea may be postponed, but the equipment should be at hand.

Clinical discussion was carried on by Dr. A. Crampton Smith, of the Department of Anesthesia, Radcliffe Infirmary, Oxford. His observations were chiefly related to poliomyelitis, which he divided into groups. The first group included patients with the bulbar type and those with associated cranial nerve disturbances affecting swallowing and glottis closure. The second group of patients had either paresis or paralysis of the muscles of respiration (while maintaining ability to swallow) and laryngeal reflexes. The third group consisted of those with a combination of the above-mentioned bulbar and muscular respiratory deficiencies. A final group consisted of those who had respiratory paralysis and who "moved over" into bulbar inadequacy. Such are "in great hazard because they may be drowned by the very machine which was to be life-saving." In all of these groups the danger is from inhaled saliva, food particles, or other invaders. When the defenses of the body which normally resist (reflexes, etc.) such enemies are deficient, the hazards to life are perilous. The discussion was carried on by several experienced surgeons and dealt with the general management of respiratory incompetence due to accident or disease. While it is interesting from a general medical standpoint, it cannot be summarized here.

I. W. VOORHEES, New York.

Nose

CHRONIC EPIDURAL ABSCESS AND CONDENSING OSTEOMYELITIS OF THE FRONTAL BONE. C. A. WHITTEN and J. B. MOYAR, J. Neurosurg. **12**:516, 1955.

Chronic epidural abscess with hyperplastic bone changes extending over a period of years without outward manifestations is not encountered frequently. Whitten and Moyar report the case of a 35-year-old woman who was admitted to the hospital with the complaints of severe headache, nausea and vomiting, and double vision. She also noted blindness in the right visual field with unsteady, staggering gait. Her past history revealed pansinusitis 21 years before admission with roentgenographic evidence of osteomyelitis of the right frontal bone. The bone was curetted, and a small amount of purulent material was removed beneath the necrotic bone. One year later roentgenograms showed chronic osteomyelitis involving the left frontal bone and orbital plates. Drainage persisted throughout the following five years but finally ceased after débridement of the necrotic tissue and bone. She was asymptomatic throughout the following 12 years, when her symptoms of headache, nausea, and vomiting appeared. Neurological examination revealed bilateral papilledema and weakness of both lateral rectus muscles, and visual field examination showed right incongruous homonymous hemianopsia. Roentgenograms of the skull revealed an extensive sclerotic reaction along the medial aspect of the left frontal bone. At operation, a large amount of extremely thickened eburnated bone was removed, with 5 cc. of purulent material beneath this thickened bone. Culture of this material revealed nonhemolytic Staphylococcus aureus. Her headaches, vomiting, and double vision subsided.

This case demonstrates the fact that an infection of low virulence in the epidural space can exist for 12 years without outward signs of its presence. This confirms the impression that cranioplasty following contaminated or infected wounds of the skull, even though done a year or more following the original removal of bone, is not without danger.

MANDEL, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

Miscellaneous

PLASMA CELL THEORY OF ANTIBODY FORMATION. HARRY P. SCHENCK, Ann. Otol. Rhin. & Laryng. **64**:109 (March) 1955.

The plasma cell appears to be the only cellular element in lymphoid tissue capable of synthesizing antibodies and gamma globulin. Lymphoid tissue is the site of antibody formation, and the lymphoid tissue of Waldeyer's ring contains all of the cellular constituents essential for the manufacture of humoral immune bodies. Lymph nodes and other lymphatic tissue appear to be the birthplace and graveyard of lymphocytes as well as the source of antibodies. The strategic position and structural modification of the lymphoid tissue of Waldeyer's ring suggest that the prime function of this tissue is the formation of antibodies and other gamma globulins.

STEELE, Kansas City, Kan.

STILBAMIDINE ISETHIONATE THERAPY OF TIC DOULOUREUX. B. WOODHALL and G. L. ODOM, J. Neurosurg. **12**:495, 1955.

The authors report the use of an intravenous solution containing 0.15 gm. of stilbamidine isethionate dissolved in 150 cc. of 5% dextrose and distilled water for the treatment of severe pain in tic douloureux. The course of therapy consisted of 10 daily injections, for a total of 1.5 gm. Fifteen of the patients were men and 26 were women, with their ages varying from 32 to 86 years. The patients developed a chemical neuropathy from the stilbamidine which relieved the pain and was associated with sensory changes over the trigeminal and upper cervical dermatomes. Of the 41 patients, 36 obtained relief from their pain for a period ranging from nine months to two years. In a small number of cases, unpredictable formication and paresthesias occurred, which may decrease the therapeutic efficacy of this drug. Nevertheless, this drug may be a valuable adjunct to the care of tic douloureux, and the beneficial results should warrant further study of stilbamidine for intravenous and oral use.

MANDEL, Philadelphia [A. M. A. ARCH. NEUROL & PSYCHIAT.].

DIFFERENTIAL DORSAL ROOT SECTION IN THE TREATMENT OF BILATERAL TRIGEMINAL NEURALGIA. B. STOOKEY, J. Neurosurg. **12**:501, 1955.

Bilateral trigeminal neuralgia occurring with or appearing after unilateral trigeminal neuralgia imposes a formidable problem of treatment. The consequences of bilateral loss of sensation and the inherent threat of damage to the motor innervation of the muscles of mastication have made the surgeons reluctant to operate.

Stookey reports four cases of bilateral trigeminal neuraliga which was treated by bilateral selective dorsal root section of the trigeminal nerve by the transtemporal approach. In each instance, the patient had had a previous episode of trigeminal neuralgia of the opposite side which had been treated by similar selective dorsal root section. Prior to the second operation, conservative treatment, consisting of alcohol injections and high doses of cyanocobalamin, had failed to alleviate the pain. Following surgery, each patient experienced freedom from pain and had no weakness of the muscles of mastication.

Stookey advocates the transtemporal approach for root section because of the ease with which the operation may be performed and the lack of complications through selective root section. He does not recommend tractotomy, since the cerebellar pathways and fibers to the masticatory muscles may be involved in high sections and the ophthalmic fibers in the descending trigeminal tract may be injured in the more caudal tractotomy.

MANDEL, Philadelphia [A. M. A. ARCH. NEUROL & PSYCHIAT.].

PROBLEMS IN EARLY DIAGNOSIS OF CARCINOMA OF THE LUNG. ARTHUR J. CRACOVANER, New York J. Med. **56**:90 (Jan. 1) 1956.

This paper was read before the Third International Congress of Broncho-Esophagology, at Lisbon, Portugal, October, 1954. Cracovaner is an ENT surgeon at the Lenox Hill and Veterans Administration Hospitals, New York. He stresses early diagnosis in carcinoma of the lung, based on team work in a group consisting of an internist, chest specialist, roentgenologist, pathologist, and bronchologist. The curability rate is still too low: 6%-8% in all patients operated on and 40% in the resectable and most favorable cases. Everyone knows of the great increase in cancer of the lung, but still we do not see the patient early enough. Procrastination of the patient who has a cough, hemoptysis, and wheeze is too often coupled with delayed diagnosis by the first physician to treat him. Any person who has the above symptoms persisting for more than three weeks should be under suspicion and needs team diagnosis. X-ray studies are, to be sure, helpful, and yet "negative findings" do not prove that cancer is absent. The bronchoscopist must observe every abnormality no matter how slight; for example, a tiny elevation of the mucosa with slight roughening or irregularity which bleeds on touching is suspicion-arousing. One must remove a piece of it by forceps or curette or wash the lung with saline and collect the washings for laboratory study. A change of color should be noted and also any narrowing of the cartilaginous rings. Moreover, one such introit is not enough. We may have to go back at several sittings for more specimens. We are leaning more and more on the pathologist, but we should not hesitate to call in the thoracic surgeon promptly.

I. W. VOORHEES, New York.

MECHANISMS INVOLVED IN ANESTHETIC DEATHS. ROBERT A. HINGSON, WILLLIAM D. HOLDEN, and ALLEN C. BARNES, New York J. Med. **56**:230 (Jan. 15) 1956.

This is a study undertaken by three Cleveland obstetricians as a "survey" of operating-room fatalities.

A quotation avers that there is "one anesthetic related death to each 1,000 anesthesias!" This may seem like a small percentage, but even one is too many! When a patient "goes bad" on the table, there is consternation, and unless every member of the team is prepared for such an event, there is likely to be chaos. Therefore, certain maxims must be set down. 1. Anesthetists must be thoroughly trained. 2. Teamwork must maintain among surgeons, anesthetists, and the entire operating-room staff, before and during surgery. 3. In readiness must be the lifesaving resuscitative equipment, such as mechanical airways, the tools for intubation, suction, and reduction of resistance to induction (especially in small children), and tables which can be readied for use of the Trendelenburg position in a moment. 4. Restorative intravenous fluids, including blood infusions, should be on hand. 5. Properly staffed recovery

rooms should be available. Table VII sets down nine causes of operating-room deaths observed in University Hospitals in Cleveland during 1945-1954. The first cause is cardiac arrest, followed by aspiration of foreign bodies (blood and vomitus), uncontrollable hemorrhage, shock, and "indeterminate causes." Ten tables and 13 references to the literature are set down.

ABSTRACTER'S COMMENT: Every surgeon will, at some moment in his career, have a death on the operating table. The law of averages will see to that. The first one will come as a blow to one's sense of pride and tend to negate his skill. Clinical hypoxia occurs in large well-staffed hospitals under favorable conditions for control. What must be happening in small, remote institutions without any set-up for O.R. emergencies? In 1933, the Society for the Prevention of Asphyxial Death was set up by Dr. Paluel J. Flagg at the New York Academy of Medicine. Its purpose is to train hospital staffs in the principles of pneumatology, to identify the causes of patients "going bad on the table," to prevent catastrophes, and to resuscitate the patients. It has had hard sledding and is still not widely enough accepted. Studies such as Hingson and his associates are making should galvanize every physician and surgeon into action. We have much of the knowledge at hand. It is little short of criminality not to apply it

I. W. VOORHEES, New York.

THE OCCURRENCE OF MALIGNANCIES IN CHILDREN. VINCENT H. HANDY and IRVING D. GOLDBERG, New York J. Med. **56**:258 (Jan. 15) 1956.

Since 1940 cancer has been a reportable disease in New York City. Statistics state that in a three-year period (1951-1953) 49% of cases under 15 years of age were discovered before the age of 5; 31% between ages 5 and 9, and only 20% were in the 10-14 group. This is rather surprising, for most of us would suppose that the number of malignancies increase with age. Perhaps they do after adulthood is reached. Table III in this study shows that leukemia, which some like to call "cancer of the blood," heads the list—114 cases under 5 years; 67 between 9 and 5; 33 between 10 and 14. ENT specialists should be warned to keep in mind the possible malignancy of very large tonsils and nasal obstruction which may not be entirely due to adenoids. Sarcoma is not too rare in the ages during which adenotonsillectomy operations are most likely to be performed. As for leukemia, its incidence has been emphasized recently in radio and in magazine articles, and it has been described as the number one killer of children. This is probably too strong a statement, but if one is impressed, it might be a good idea to know the blood picture of every little patient who comes in for an adenotonsillectomy operation. If one insists on knowing coagulation and clotting time, the complete blood cell count could be done as routine. Surgery on a child with leukemia is an ordeal into which none of us would care to venture unless under great compulsion.

I. W. VOORHEES, New York.

CLINICAL ANESTHESIA CONFERENCE: DEATH FROM BRONCHIAL ASTHMA. BARNET A. GREENE, New York J. Med. **56**:273 (Jan. 15) 1956.

Death due to bronchial asthma in a woman under spinal anesthesia carries a condemnatory note which is rare in medical reporting. The anesthesiologist failed to prevent this death by omitting the application of medical principles in the practice of anesthesiology in an asthmatic patient. A white woman, aged 31, was admitted for severe low abdominal pain, in the evening. Next morning, surgery was undertaken for the removal of "a mildly inflamed appendix." A part of each ovary was also removed. Evidently the anesthetist favored spinal to lung anesthesia, knowing of the symptoms, signs, and history of bronchial asthma. It is unnecessary to recite the entire anamnesis here, but this was not a "table case," since death took place after the patient's return to bed. Autopsy showed the entire bronchial tree on both sides to be filled with a thick gelatinous substance. On this ground the anesthetist is rebuked for not freeing the airways by suction removal. It is also stated that this was no excessively urgent condition. The patient could have been prepared for some hours preceding surgery with the use of corticotropin (ACTH) or adrenocortical agents or even an antihistamine, aminophylline, and epinephrine before, during, and after anesthesia.

ABSTRACTER'S COMMENT: Again we have a case where direct intubation with suction might have saved a life. Asphyxial death from "drowning in his own secretions" is a phrase which belongs to backwoods medicine. It is doubtful whether it should occur in these days of skillful bronchoscopy.

I. W. VOORHEES, New York.

FUNDAMENTALS OF MODERN ALLERGY. HARRY M. SHILKRET, New York J. Med. **56**:420 (Feb. 1) 1956.

This in one of a series of articles sponsored by the New York Allergy Society. Dr. Shilkret works in the Allergy Clinic, City Hospital. In this article he mentions specifically allergies acquired in beauty parlors due to the inhalation of orris powder, henna dye, quince-seed oil, and gums. Hat makers are exposed to rabbit dander and bleaching agents (banana oil and oxalic acid). Upholsters inhale dust from mattress fillings (kapok, etc.). Bronchial asthma is a health hazard in many occupations. One needs to ask whether the person had asthma before employment or contracted it in his work. Compensation examiners must know such details. How to protect the worker becomes a sort of engineering matter. Nasal filters are objected to by many, who "cannot be bothered." Therapy through specific hyposensitization and the use of drugs to combat symptoms, chiefly for relief, are to be considered. Serious cases, those who are fairly disabled by allergies no matter what measures are taken, should change to another occupation.

ABSTRACTER'S COMMENT: Nothing is said here concerning the so-called psychosomatic causes of allergy, but it is possible to have an organism highly sensitized by an environment outside of a work room (unhappy home environment, etc.) which would intensify the action of baneful material agents. Here is a subject which calls for a "broad-spectrum" view, as current parlance goes.

I. W. VOORHEES, New York.

USE OF HORMONES IN THE TREATMENT OF ALLERGIC DISEASES. EMANUEL SCHWARTZ, New York J. Med. **56**:570 (Feb. 15) 1956.

This is another in the series of special articles written under the aegis of the New York Allergy Society, Dr. Samuel J. Prigal, Editor. Hormone therapy is no substitute for the accepted methods of treatment; the old prophylaxis—avoidance of causes, offending agents, and specific hyposensitization, etc.—must still be used. However, corticosteroids plus antibiotics for from two to seven days for asthmatic attacks ushered in by acute respiratory infections may diminish them in intensity and prevent status asthmaticus. These "newer drugs" are adjuvant to epinephrine, iodides, barbiturates, intravenous dextrose, etc. After relief has been obtained one may cut down and eventually eliminate the hormones. However, management calls for an intimate knowledge of the action of hormones in allergic states and is scarcely open to experimentation. "Corticotropin, cortisone, hydrocortisone, prednisone, predisolone dramatically suppress many of the clinical manifestations of allergic diseases, and are the most effective agents available at present for symptomatic relief in carefully selected cases, after the usual therapeutic measures . . . have failed." A figure illustrates the physiologic regulation of adrenocortical secretion, dramatically and graphically adapted and modified by the well-known medical illustrator, Dr. F. Netter. In addition, some 30 references are set down as a background for this essay.

I. W. VOORHEES, New York.

News and Comment

ANNOUNCEMENTS

The Ninth Annual Conference on Electrical Techniques in Medicine and Biology.—The Ninth Annual Conference on Electrical Techniques in Medicine and Biology will be held at the Governor Clinton Hotel in New York City on Nov. 7, 8, and 9, 1956.

The Conference is sponsored by the American Institute of Electrical Engineers, the Institute of Radio Engineers, and the Instrument Society of America. In addition to the technical sessions, there will be field trips to installations of interest to those attending.

Titles and brief abstracts for proffered papers may be submitted to the Conference Chairman, E. Dale Trout, General Electric Company, X-Ray Department, Milwaukee 1, Wis.

Books

Précis d'audiométrie clinique. By Michel Portmann and Claudine Portmann. Price, 2500 francs. Pp. 432, with 138 illustrations. Masson & Cie, 120 boulevard Saint-Germain, Paris, 6e, 1954.

This book may be aptly termed the credo of the Bordeaux School of Clinical Audiology. It emphasizes the practical side of the latest diagnostic and therapeutic methods used in the treatment of hearing disorders at the University of Bordeaux. Each type of hearing problem is preceded by a theoretical introduction. The material presented is succinct, yet thorough, and is designed to aid the clinician with or without elaborate equipment. Of particular interest is the authors' extensive analysis of the etiology of conductive, nerve-type, and mixed deafness, accompanied by excellent descriptions of testing procedures with audiograms of actual tests. The suggestions on how to select a suitable prosthetic device are very helpful. There is cogent evidence of the value of audiometry in legal and industrial medicine. The chapters devoted to deafness in children contain informative evaluations of tests which take into account both the mental and chronological ages of the patients. The book's usefulness is limited, however, since it is intended for readers of French.

A Stereoscopic Atlas of Human Anatomy. Section III: The Upper Extremity. By David L. Bassett, M.D. Stereoscopic color photographs by William B. Gruber. Price, $22.50. Pp. 400 (spiral bound), with 196 stereoscope views on 28 reels in 3 volumes. The Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1955.

This important contribution for teaching anatomy with the use of stereoscopic 35 millimeter still films now includes volumes on the upper extremity. The 28 reels consist of beautifully taken stereoscopic films depicting anatomical dissections of the upper extremity. The films are for projection, and each film is accompanied by a detailed sketch showing the names of the anatomical parts.

Previous sections have dealt with the central nervous system and the anatomy of the head and neck. All these volumes are certainly worth the attention of everyone who performs surgery, and teaches anatomy.

Functional Otology: The Practice of Audiology. By Morris F. Heller, M.D., with Bernard M. Anderman, M.A., and Ellis E. Singer, M.A. Price, $5.50. Pp. 240, with 31 illustrations. Springer Publishing Company, Inc., 44 E. 23rd St., New York 10, 1955.

In this book of 240 pages Heller has more than accomplished his intent of coordinating information on audiology. With few exceptions, the information in the book respresents the best present-day thinking. Of course, the subjects are covered only superficially and serve more to supply a good perspective rather than a working background. Heller has been influenced to a great degree by his work in a large audiology center. For example, he describes as a recommended audiology center one of such a comprehensive nature that the reader may be led to believe an excellent audiology center cannot function without a staff of some 14 professionals, including a social worker, consultants, and audiologists who have the highest available certification. He is describing an ideal situation, but the same results for the patient can often be obtained with only a few devoted and competent specialists.

The book is a much needed and worth-while contribution to otology.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. D. S. Gorrell, 110 Medical Arts Building, Calgary, Alta.

Secretary: Dr. G. Arnold Henry, 328 Medical Arts Building, Toronto.

Place: Chateau Frontenac, Quebec, Canada. Time: June 6-7, 1956.

International Bronchoesophagological Society

President: Dr. Antonio da Costa Quinta, Lisbon, Portugal.

Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

Meeting: Fourth International Congress of Bronchoesophagology.

President of Congress: Dr. Antonio Carrascosa.

International Congress of Audiology

Place: Leyden, Netherlands.

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

International Course in Paedo-Audiology

Place: Groningen, Netherlands.

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Juan Manuel Tato, Azcuenaga 235, Buenos Aires, Argentina.

Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

Sixth International Congress of Otolaryngology.

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.

General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.

Place: Hotel Statler, Washington, D. C. Time: May 5-10, 1957.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. John R. Lindsay, Chicago.

Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.

Place: Chicago. Time: June 11-15, 1956.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Algernon B. Reese, 73 E. 71st St., New York 21.

Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave., Bldg., Rochester, Minn.

American Board of Otolaryngology

President: Dr. Frederick T, Hill, Professional Building, Waterville, Maine.

Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

Place: Sheraton-Mount Royal Hotel, Montreal, Canada. Time: May 6-11, 1956.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Daniel S. Cunning, 115 E. 65th St., New York 21.

Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.

Place: Sheraton-Mount Royal Hotel, Montreal, Canada. Time: May 15-16, 1956 (afternoons only).

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. Bernard J. McMahon, 8230 Forsyth Blvd., Clayton 24, Mo.

Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.

Place: Seigniory Club, Montreal, Canada. Time: May 13-14, 1956.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Dean M. Lierle, Iowa City.

Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

Place: Sheraton-Mount Royal Hotel, Montreal, Canada. Time: May 15-17, 1956 (mornings only).

SECTIONS:

Eastern.—Chairman: Dr. John A. Murtagh, Hanover, N. H.

Southern.—Chairman: Dr. J. M. Robison, Houston, Texas.

Middle.—Chairman: Dr. William H. Craddock, Cincinnati.

Western.—Chairman: Dr. David D. De Weese, Portland, Ore.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. William J. McNally, 1509 Sherbrooke St. West, Montreal 25, Canada.

Secretary-Treasurer: Dr. Lawrence R. Boies, 90 S. Ninth St., Minneapolis 2.

Place: Seigniory Club, Montreal, Canada. Time: May 11-12, 1956.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

President: Dr. Joseph Gilbert.

Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. Dean McAllister Lierle, University Hospital, Iowa City.

Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.

Place: Palmer House, Chicago.

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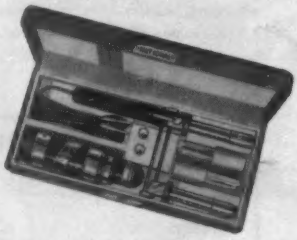

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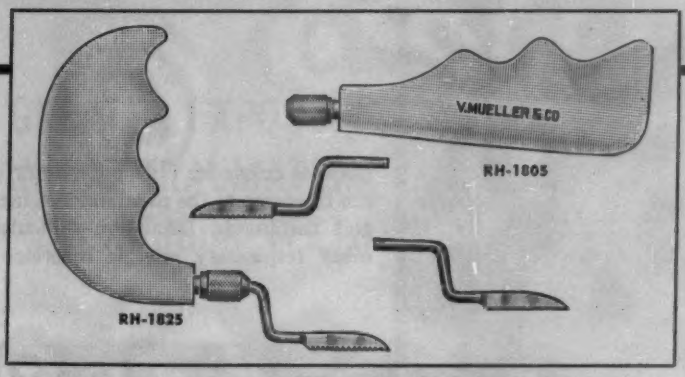

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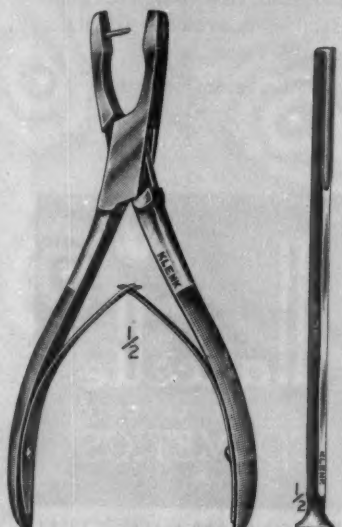

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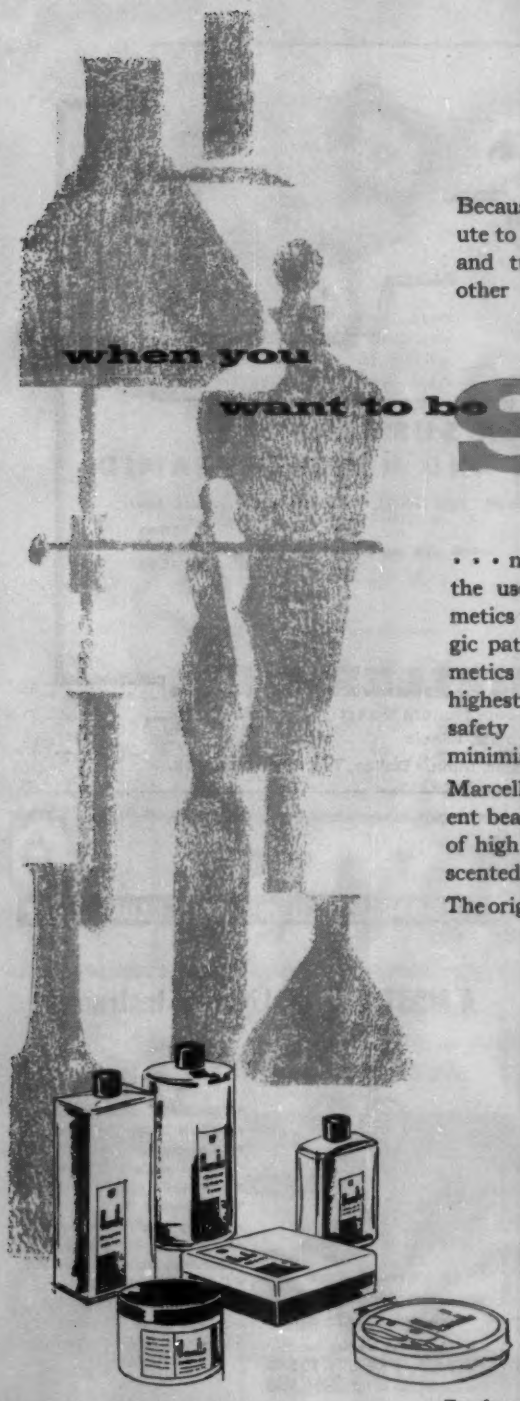

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